Medical Library
MAY 16 1942

VOL. IV. Nos. 1 & 2 JANUARY & APRIL, 1942

BRITISH HEART JOURNAL

FOR THE STUDY OF
CARDIOLOGY AND THE CIRCULATION

EDITORS
MAURICE CAMPBELL
D. EVAN BEDFORD

EDITORIAL BOARD

CRIGHTON BRAMWELL, MANCHESTER
BOYD CAMPBELL, BELFAST
JOHN COWAN, GLASGOW
FRANCIS R. FRASER, LONDON
A. G. GIBSON, OXFORD
JOHN HAY, LIVERPOOL
SIR THOMAS LEWIS, LONDON
HENRY MOORE, DUBLIN
JOHN PARKINSON, LONDON
W. T. RITCHIE, EDINBURGH
K. D. WILKINSON, BIRMINGHAM
EDITOR OF BRITISH MEDICAL JOURNAL

APPOINTED BY
THE CARDIAC SOCIETY OF GREAT BRITAIN AND IRELAND

LONDON
BRITISH MEDICAL ASSOCIATION
TAVISTOCK SQUARE, W.C.1

PUBLISHED QUARTERLY SUBSCRIPTION 25/- PER ANNUM. U.S.A. $6.00

CONTENTS

PAGE

Two Cases of Digitalis Poisoning. By K. DOUGLAS WILKINSON . 1

Congenital Heart Block. By WILLIAM STEIN and J. S. UHR . . 7

Congenital Pulmonary Stenosis with left ventricular enlargement associated with atrial septal defect. By PAUL WOOD . . 11

Infarction of the Cardiac Auricles (Atria). By E. H. CUSHING, H. S. FEIL, E. J. STANTON, and W. B. WARTMAN . 17

Prognosis of Right Bundle Branch Block. By GEORGE A. PERERA, SAMUEL A. LEVINE, and HERMAN ERLANGER 35

Dr. Samuel Johnson: The Medical History as recorded by James Boswell. By TERENCE EAST 43

Inversion of T Waves after Long Paroxysms of Tachycardia. By MAURICE CAMPBELL 49

TWO CASES OF DIGITALIS POISONING

BY

K. DOUGLAS WILKINSON

From the Birmingham United Hospital

Received October 14, 1941

In 1785 Dr. William Withering of Birmingham wrote, " The Foxglove when given in very large and quickly repeated doses, occasions sickness, vomiting, purging, giddiness, confused vision, objects appearing green or yellow; increased secretion of urine, with frequent motions to part with it, and sometimes inability to retain it; slow pulse, even as slow as 35 in a minute, cold sweats, convulsions, syncope, death. I am doubtful whether it does not sometimes excite a copious flow of saliva.

" When given in a less violent manner, it produces most of these effects in a lower degree; and it is curious to observe that the sickness with a certain dose of the medicine does not take place for many hours after its exhibition has been discontinued; that the flow of urine will often precede, sometimes accompany, frequently follow the sickness at the distance of some days, and not unfrequently be checked by it. The sickness thus excited is extremely different from that occasioned by any other medicine; it is peculiarly distressing to the patient; it ceases, it recurs again as violent as before; and thus it will continue to recur for three or four days, at distant and more distant intervals.

" At first I thought it necessary to bring on and continue the sickness, in order to ensure the diuretic effects. Therefore my patients were ordered to persist until the nausea came on, and then to stop. But it soon appeared that the diuretic effects would often take place first, and sometimes be checked when the sickness or a purging supervened.

" The direction was therefore enlarged thus—Continue the medicine until the urine flows, or the sickness or purging take place. I found myself safe under this regulation for two or three years; but at length cases occurred in which the pulse would be retarded to an alarming degree, without any other preceding effect.

" The directions therefore required an additional attention to the state of the pulse, and it was moreover of consequence not to repeat the doses too quickly, but to allow sufficient time for the effects of each to take place, as it was found very possible to pour in an injurious quantity of the medicine before any of the signals for forbearance appeared.

" Let the medicine therefore be given in the doses, and at the intervals mentioned above:—let it be continued until it either acts upon the kidneys, the

stomach, the pulse, or the bowels; let it be stopped upon the first appearance of any of these effects, and I will maintain that the patient will not suffer from its exhibition, nor the practitioner be disappointed in any reasonable expectation.

" From some cases, which have occurred in the course of the present year, I am disposed to believe that the digitalis may be given in small doses, viz., two or three grains a day, so as gradually to remove a dropsy, without any other than mild diuretic effects, and without any interruption to its use until the cure be completed.

" If inadvertently the doses of the Foxglove should be prescribed too largely, exhibited too rapidly, or urged to too great a length, the knowledge of a remedy to counteract its effects would be a desirable thing."

Withering used the Foxglove to promote diuresis; his dosage was so admirable that it remains to-day the standard in general use. His directions were so simple that they cannot be improved, and he was well acquainted with digitalis poisoning, yet did not succeed in impressing upon his contemporaries the importance of the cautious doses he recommended. It is said that Dr. J. Lettson employed the medicine so incautiously that he killed several patients, and certainly many physicians regarded the digitalis as a dangerous drug.

Since Withering wrote we have learned of the active principles of digitalis—the glucosides—and of their action upon the myocardium for which they have a peculiar chemical affinity. Now we use digitalis chiefly in auricular fibrillation with the object of limiting the frequency of the ventricular contraction, prolonging the diastolic pauses, and so improving the nutrition of the heart.

These effects result from the blocking of impulses from the auricles travelling in the A-V bundle. Adequate doses of digitalis may cause complete heart block, either in auricular fibrillation or in sinus rhythm, and high grades of auriculo-ventricular dissociation can be produced relatively easily in hearts that have been damaged by rheumatism. The chemical affinity of digitalis glucosides for the myocardium is much greater than that of strophanthin, so that the effects of digitalis are much more lasting, poisoning may persist for a long time after the discontinuance of the drug, and as yet we have not found the antidote for which Withering hoped.

Even to-day digitalis is still used in dangerous doses, and in the hope of producing impossible results in unsuitable cases, so that poisoning is not uncommon. Marked slowing of the pulse rate and coupling as a result of the appearance of ventricular extrasystoles are the first two cardiac signs of digitalis intoxication. In the two cases here reported there was also widening of the QRS complexes.

First Case

Winifred B., aged 27, gave a history of rheumatic fever at the age of 10, goitre during adolescence, appendicectomy at the age of 18, but otherwise had enjoyed good health until July 1940. Then six-and-a-half months pregnant with her second child, she began to have attacks of paroxysmal tachycardia.

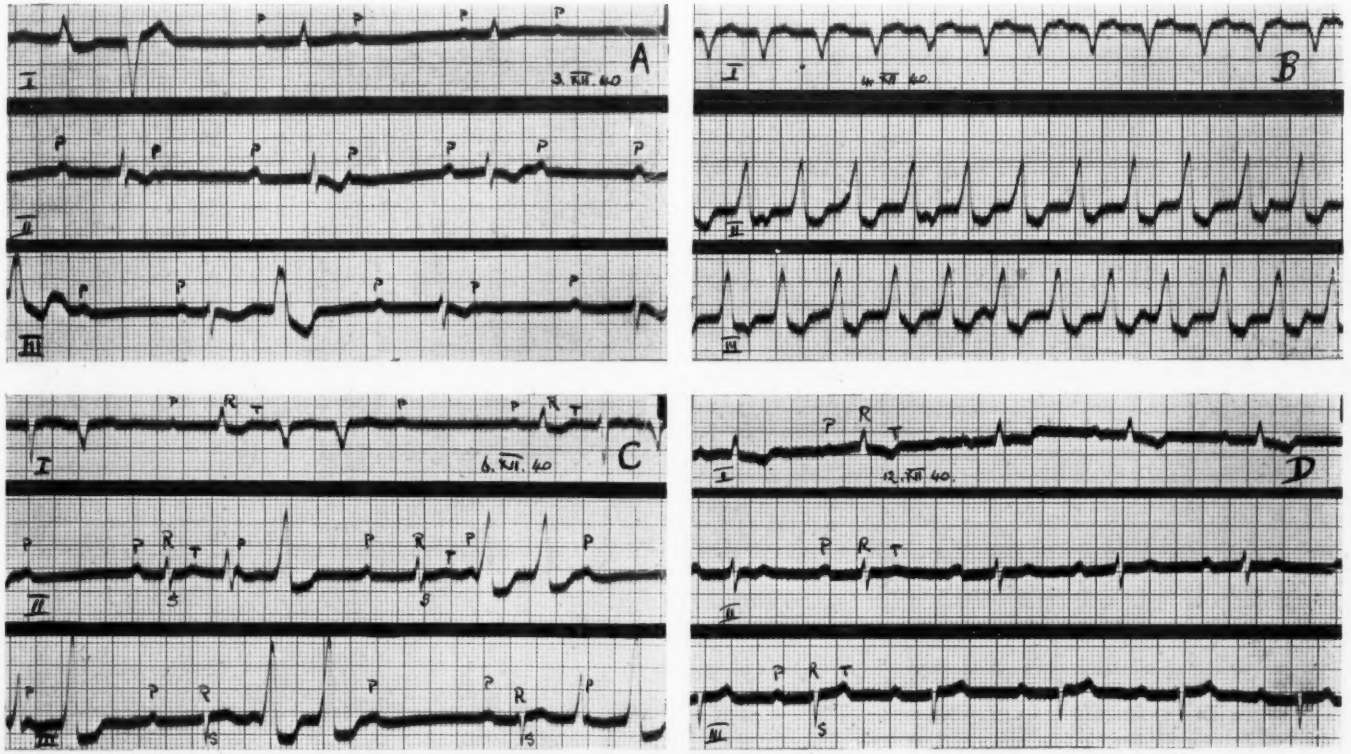


FIG. 1.—(A) Case 1; 3/12/40.—Complete A-V dissociation (auricular rate of 85 and ventricular rate of 34) with ventricular extrasystoles.
(B) Case 1; 4/12/40.—Paroxysmal ventricular tachycardia, rate about 150.
(C) Case 1; 6/12/40.—Ventricular extrasystoles, often in pains; complete A-V dissociation still present.
(D) Case 1; 9/12/40.—Partial heart block still present, 12 days after the last dose of digitalis.

She was admitted to the Maternity Hospital, where she had a normal delivery, after which the attacks ceased; but a month or so later started again, and on November 27 she was ordered two grains of digitalis leaf every three hours. This she continued to take conscientiously until the day of admission, in spite of the persistence of the attacks of palpitation, nausea, vomiting, and disturbed vision.

When admitted to hospital, under my colleague Professor L. G. Parsons, on December 2, 1940, she was most gravely ill, but was not breathless nor cyanosed. The cardiac impulse could not be felt, the pulse was of small volume, infrequent (less than 50 per minute), and irregular with coupled beats. The tongue was furred and vomiting was frequent; in fact nausea almost prevented her taking anything by mouth and she had to be given rectal saline.

The electrocardiogram taken the day after admission showed a sinus rate of about 85, the P waves being small but quite easily identified; the ventricular rate was variable, about 34 per minute, and there were ventricular extrasystoles of constant form. There was complete auriculo-ventricular dissociation (Fig. 1A).

The paroxysms of ventricular tachycardia, for which the heroic doses of digitalis folia were prescribed in this case, were not modified by the large doses, for the attacks continued, and Fig. 1B shows such an attack on December 4, on which day her heart suddenly changed from an irregular rate of about 40 per minute to a regular rate of nearly 150; this attack only lasted about half an hour, and ceased as abruptly as it began, but she had several subsequent attacks.

Sometimes the ventricular extrasystoles occurred in pairs, which varied in form and appeared to arise from different foci; they were separated by a constant interval. This condition is well seen in Fig. 1C, where each normal ventricular complex is followed by two ventricular extrasystoles. Complete heart block appears to be present on this date also.

On December 8 she was given quinidine sulphate in doses of three grains every two hours for five doses, and this led to an improvement in that the paroxysms of tachycardia ceased; but the persistence of digitalis block is well seen in the record taken on December 12, where the rate has become regular at 64 per minute; the P–R interval was still 0·32 sec., although the digitalis had been stopped twelve days earlier (Fig. 1D). Subsequently she gradually improved and was discharged as cured on January 30, 1941.

Second Case

But adequate doses of digitalis can produce even greater changes in the electrocardiogram than those shown in the first case.

The second patient, William H., aged 57, was admitted under Professor W. H. Wynn on March 21, 1941.

He also gave a history of acute rheumatism as a child, but served in the Army from 1914 to 1920. He had appendicitis in 1933, and in 1939 was admitted to hospital with congestive heart failure. He recovered and returned

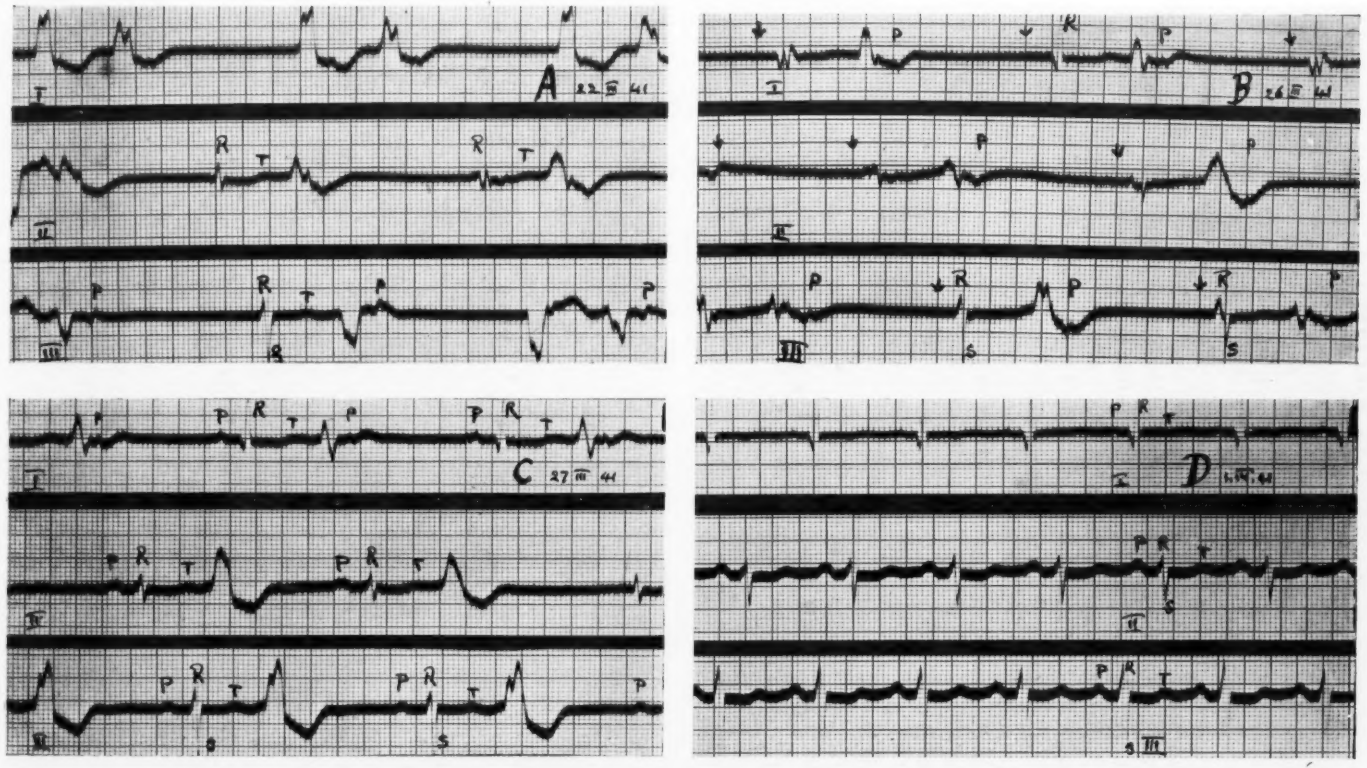


FIG. 2.—(A) Case 2; 22/3/41.—Coupled beats with most complexes of the bundle branch block type (see text).
(B) Case 2; 26/3/41.—Coupled beats with more normal ventricular complexes (see text).
(C) Case 2; 27/3/41.—Coupled beats still present, but P waves now obvious with P–R interval of 0·24 sec.
(D) Case 2; 1/4/41.—Regular sinus rhythm with a P–R interval of 0·20 sec.

to work. In December 1940, he became distressed by increasing dyspnœa and began to have some œdema of the legs. On admission he was plethoric, œdematous, cyanosed, and orthopnœic, and weighed 12 stone 7 pounds (the degree of œdema was shown by the fact that he lost 3 stone in sixteen days). The pulse was infrequent (about 50 per minute), of poor volume, and showed coupled beats. The blood pressure was 175/110. The cardiac impulse was five inches from the mid-line in the seventh left interspace; the heart sounds were distant and he was extremely ill. Before admission he had been taking digitalis, but the dosage was unknown.

The record of March 22, 1941, shows only two ventricular complexes (one in lead II and one in lead III) that are even approximately normal; the remainder appear fairly typical of bundle branch block of the common type, being wide (0·16 sec.), notched, and having T waves directed opposite to the initial deflection (Fig. 2A). This record also shows coupled beats, while the auricular complexes cannot be identified with certainty, but since the ventricular rate is about 30 per minute it seems probable that complete auriculo-ventricular dissociation was present.

Four days later a similar record was obtained, showing the nearly regular ventricle with a frequency of 30, each beat being followed by an extrasystole (Fig. 2B). Here again the ventricular complexes, both the initial complexes and the extrasystoles, show considerable variation in form, although they are separated by an almost constant interval of 0·6 sec., and the auricular complexes can only be identified occasionally: but a day later (Fig. 2C), P waves can be seen, the P–R interval being about 0·24 sec. and constant, and the ventricular complexes much less abnormal; coupling is still present, and the extrasystole is of an abnormal type.

From March 21 to March 28 the pulse varied from 70 to 32 per minute; during the second week it rose to an average of 80, and thereafter was above 80 and regular. On April 1, his electrocardiogram (Fig. 2D) showed a regular sinus rhythm of 77 with a P–R interval of 0·20 sec., the disappearance of extrasystoles, and a constant QRS deflection: S was still deep in leads II and III, but QRS now measured at most 0·09 sec., and T although rather flat was upright. He was discharged much improved on May 24, 1941.

Summary and Conclusions

Two cases of digitalis poisoning in patients with sinus rhythm are recorded. Each showed : (1) complete auriculo-ventricular dissociation; (2) coupled beats; and (3) marked abnormalities in the QRS-T complexes.

Recovery was slow and some degree of heart block persisted for twelve days after the digitalis was discontinued. One case showed bundle branch block as well as auriculo-ventricular block.

CONGENITAL HEART BLOCK:
REPORT OF A CASE

BY

WILLIAM STEIN AND J. S. UHR

From St. Peter's General Hospital, New Brunswick, N.J., U.S.A.

Received October 20, 1941

The comparative rarity of congenital heart block as recorded in medical journals warrants the addition of another case to this collection. Up to 1938 approximately 55 cases have been described.

There is no doubt that one of the reasons for not discovering these cases early in childhood is that signs and symptoms are usually absent or only minimal, and that the heart rate is inclined to be more rapid than in cases with acquired complete heart block (Campbell and Suzman, 1934). It is only when some other medical condition brings such a person to a physician that the heart block may be discovered. Sometimes this person may be examined by several competent doctors before the defect is accidentally stumbled upon. This is illustrated by the presentation of the following case.

A white, female child, aged 3 years, was brought to the hospital in February 1940 with the complaint of a purulent discharge from the right ear of one year's duration. This had started four months after an uneventful convalescence from measles, when she had been treated by a physician who made no comment about her heart. Two previous attacks of acute bronchitis also requiring the attention of a physician had caused no comment relative to her heart. Otherwise she had always been a normal-looking, well-nourished, and healthy, active youngster, without any cardiac signs or symptoms. She was a full-term baby, delivered normally, and experienced no difficulties in her babyhood. A clinical diagnosis of right acute mastoiditis was made. There were no other findings at this time. The intern made a note that he heard no murmurs over the praecordium but did not record the pulse or cardiac rate. The nurse's chart showed a pulse rate of 80. She had an operation on the same day.

During the entire time of operation the anæsthetist noted the pulse rate as being 56, and brought it to the surgeon's attention. Her condition despite this rate was good. The oto-laryngologist commented on the possibility of the low rate being due to an intradural abscess, and after completing a simple mastoidectomy asked for a neurological consultation.

Two days later the neurologist reported that he found no neurological signs, and suggested that a pædiatrician should look for some congenital anomaly because of the bradycardia.

It was then that a pædiatrician (J. S. U.) first saw her and noted " an extensive bradycardia and a low musical murmur, systolic in time, heard all over the præcordium, but loudest at the apex and fourth left intercostal space near the sternum." This was thought to be a murmur that could be accounted for most logically on the basis of a patent interventricular septum being present, inasmuch as after it had been heard it had always been present and had never varied in intensity, quality, or location.

An electrocardiogram revealed an auricular rate of 120, a ventricular rate of 70, complete heart block, a tendency to left axis deviation, and a diphasic T wave in lead IV F (Fig. 1).

Fluoroscopic and radiographic examination of the heart revealed enlargement of both the right and left ventricles and left auricle (Fig. 2). The lung

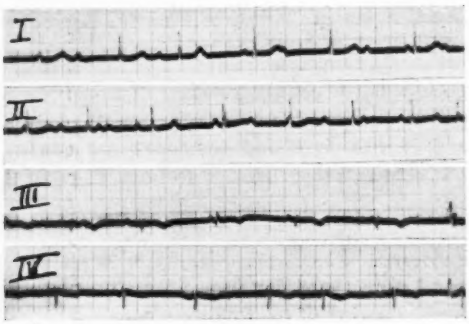


FIG. 1.—Electrocardiogram showing complete heart block (A, 120; V, 70).

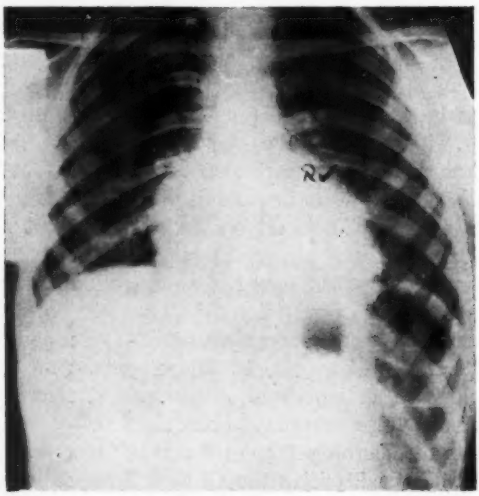


FIG. 2.—Radiogram of heart showing enlargement of the right and left ventricles.

fields were clear. Barium in the œsophagus in the right (I) oblique view showed an indentation of the œsophagus from a moderately enlarged left auricle.

Other laboratory findings were as follows. There was no abnormality of the urine except for a faint trace of albumin. Complete blood count: haemoglobin, 78 per cent; red blood corpuscles, 4,560,000; white blood corpuscles, 9050, with a normal differential count.

The rectal temperature on admission was 99·6° F. and varied from 100 to 99 until the patient was discharged.

The nurse's chart recorded a pulse average of from 56 to 52 during the patient's stay in hospital. The respirations averaged from 24 to 28 per minute.

The patient responded well to the operation and was discharged after two weeks, returning to the out-patient department for mastoid surgical dressings and follow-up in the pædiatric clinic. Two months later another electrocardiogram during one of her visits to the out-patient department again showed complete heart block.

Her convalescence from the right mastoidectomy was uneventful.

Discussion

Complete heart block represents an inability of the stimulating impulses originating in the sino-auricular node to pass through the auriculo-ventricular node and the main bundle of His, so that the auricles beat at their own rate while the ventricles contract at the rate governed by the auriculo-ventricular node that has taken over the function of pacemaker, which is usually 30 to 50 beats per minute, or if conduction from the latter is impaired also, then at their own ventricular rate, which is usually below 40 beats per minute. This heart block may be either congenital, which is rare, or acquired, which is more usual.

Yater (1929) reviewed 30 cases of congenital heart block reported up to that time, and established certain criteria that would permit one to classify a case as being congenital. These were the following five points:

1. Electrocardiographic evidence of the block existing.

2. A slow pulse that had been found present at an early age and had continued to be present.

3. The absence of any history suggestive of an infection that might have produced the block, as diphtheria, congenital or early acquired syphilis, rheumatic fever, or chorea.

4. A history of any one of the following: syncopal attacks, fainting spells (explained on the basis of the Adams-Stokes syndrome), vertigo, headaches, nausea and vomiting after unusual exertion, convulsions, dyspnœa, and/or cyanosis. (These findings may or may not be present and are not absolutely requisite for the criteria, but if present add further evidence.)

5. The presence of a congenital heart lesion, namely, patent interventricular septum (the latter at times being subject to an acquired endo-myocarditis, quite infrequent in this instance as far as the septum itself is concerned).

While theoretically it might be possible to explain the heart block on a prenatal myocarditis or syphilis involving the bundle of His or a developmental

defect affecting the bundle of His, these are most unlikely to occur for practical considerations.

This case fits the criteria of Yater (1929).

The ætiology of congenital heart block is organic and is based on the pathological presence of a patent interventricular septum. Such septal defects, both large and small, are a common deformity and are found relatively frequently. Why they occur clinically without the presence of heart block so often, is odd, but has been explained correctly as follows : The usual sight of the interventricular defect is anterior to the pars membranacea while the A-V bundle lies behind it (Leech, 1930). Inasmuch as the ventricular complexes in the electrocardiogram of complete heart block are normal, it follows that the lesion responsible for the block must be in the course of the main bundle of His, above the bifurcation close to the septum (Lampard, 1928).

The reason for the almost constant preservation of the muscular connection between the auricles and ventricles probably lies in the fact that the special bundle appears in the fifth week of fœtal life, whereas the membranous separation between auricles and ventricles take form between seven and ten weeks. The bundle is preserved between the posterior endocardial cushion and the posterior portion of the annular fibrosis (Yater, Leaman, and Cornell, 1934; Moll, 1912; and Tandler, 1913).

It is only when the A-V node or main bundle of His is caught in the congenital anomalous development of the septum, or is caught in the excessive formation of fibrous tissue of the membranous portion of the septum interfering with the continuity of the bundle, that heart block occurs (Aitken, 1932).

The prognosis in general is guarded. A few cases have reached mature adult life. It must be remembered that a patent interventricular septum is associated often with other congenital cardiac anomalies, the most frequent being the tetralogy of Fallot. These anomalies are overshadowed by the dangers of an engrafted endocarditis. If the concomitant anomalies are small and do not limit the functional capacity of the heart permitting the subject to survive early life, there is no reason to consider the prognosis unfavourable, barring an unlooked-for bacterial endocarditis.

References

Aitken, J. K. (1932). *Lancet*, **2**, 24.
Campbell, M., and Suzman, S. S. (1934). *Amer. Heart J.*, **9**, 304.
Lampard, M. E. (1928). *Arch. Dis. Childh.*, **3**, 212.
Leech, C. B. (1930). *Amer. J. Dis. Child.*, **39**, 131.
Moll, F. P. (1912). *Amer. J. Anat.*, **13**, 249.
Tandler, J. *Anatomie des Herzens, Jena*, Gustav Fischer, 1913, p. 313.
Yater, W. M. (1939). *Amer. J. Dis. Child.*, **38**, 112.
Yater, W. M., Leaman, W. G., and Cornell, V. H. (1934). *J. Amer. med. Ass.*, **102**, 1,660.

CONGENITAL PULMONARY STENOSIS

WITH LEFT VENTRICULAR ENLARGEMENT ASSOCIATED WITH ATRIAL SEPTAL DEFECT

BY

PAUL WOOD

From the Department of Medicine, British Postgraduate Medical School, Hammersmith Hospital, London

Received December 6, 1941

Observations on the morbid anatomy of congenital heart disease are in advance of clinical studies, and although we have been supplied by pathologists with much information about the meaning and incidence of the various single or combined developmental abnormalities of the heart, such knowledge is of practical value to the clinician only when he can recognize them during life. Certain well defined, single or combined, lesions may now be easily diagnosed at the bedside, especially with the aid of X-ray and electrocardiographic examination. It is the object of this report to present the clinical features of another congenital syndrome which, if not a freak, may prove recognizable at the bedside.

It is well known that marked pulmonary stenosis, as part of the tetralogy of Fallot or otherwise, causes enlargement of the right ventricle and deviation of the electrical axis of the heart to the right. It is also well known that patency of the interauricular septum produces progressive enlargement of the right ventricle, and right axis deviation or even right bundle branch block. It is interesting, therefore, that a combination of these two lesions may apparently result in left ventricular enlargement and left axis deviation. These effects are illustrated by the following case.

Description of Case

A girl, aged 6, was first seen by me in April, 1935. She had been cyanosed from birth, and had never been able to walk. She did not sit up until she was two, and since then had only been able to shuffle about on her buttocks. Her mental development was normal at first, for she began to talk when she was one, and spoke short sentences at one and a half. Because of her legs she had to be carried about, and it was soon noticed that if she were held upright suddenly, or if she exerted herself too strenuously, she was subject to fainting attacks, in which she turned deep blue, gasped for breath, and finally lost consciousness. Recovery occurred in about a minute, and there were no convulsions. From the age of four she also suffered from severe right-sided headaches, which caused her to scream for an hour or so, and of which she had no recollection. During the fortnight preceding admission to hospital, her speech became slurred and difficult to understand, and excessive salivation was bothersome.

Her parents were healthy, and there were six other children, alive and well.

Examination revealed a very cyanosed child with clubbing of the fingers and toes

FIG. 1.—Clubbed fingers.

(Fig. 1). She was in no distress, and there was no dyspnœa. The temperature was normal, the pulse rate 100 beats per minute, and the respiration rate 20 per minute. There was no focal or general infection.

Cardiovascular system. The heart was central in position, a diffuse cardiac impulse being felt as much to the right as to the left of the sternum. There were no thrills, but there was a loud and significant systolic bruit, heard rather better to the right than to the left of the mid-line, and of maximum intensity at the level of the second and third interchondral spaces. It seemed to be conducted into the vessels of the neck. The second heart sound at the base was sharp and clear, and could be heard equally well on either side of the sternum. The heart sounds were not remarkable elsewhere.

The rhythm was normal, the cervical veins were not engorged, the liver was impalpable, there was no œdema, and there was no evidence of pulmonary congestion. The blood pressure varied between 125/90 and 110/85. All the main arteries of the limbs were normal to palpation.

An electrocardiogram (Fig. 2) showed left axis deviation, and P waves which were split, up to 0·10–0·12 sec. in duration, prominent in lead I, and inverted in lead III.

The skiagram (Fig. 3) and radioscopy revealed marked prominence of the ascending aorta, left ventricular dominance, and displacement of the whole heart to the right.

A blood count showed 10,400,000 red cells per c.mm., hæmoglobin 108 per cent, white cells 3400, 39 per cent of which were polymorphonuclear cells.

Central nervous system. There was a combination of left hemiplegia and paraplegia: there was weakness of the left side of the face, the palate was drawn up to the right, the tongue was protruded to the left, the left arm was weak, and both legs were spastic, with exaggerated tendon jerks and bilateral extensor plantar responses. There was no sensory loss.

Apart from the cardiovascular and nervous systems there were no other abnormalities, except a palpable spleen.

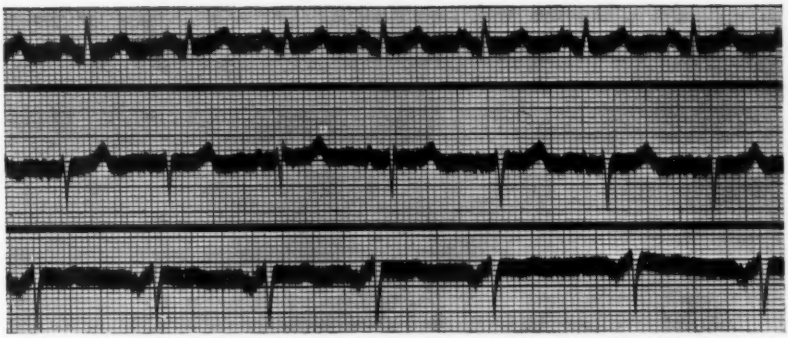


FIG. 2.—Electrocardiogram of case, showing left axis deviation.

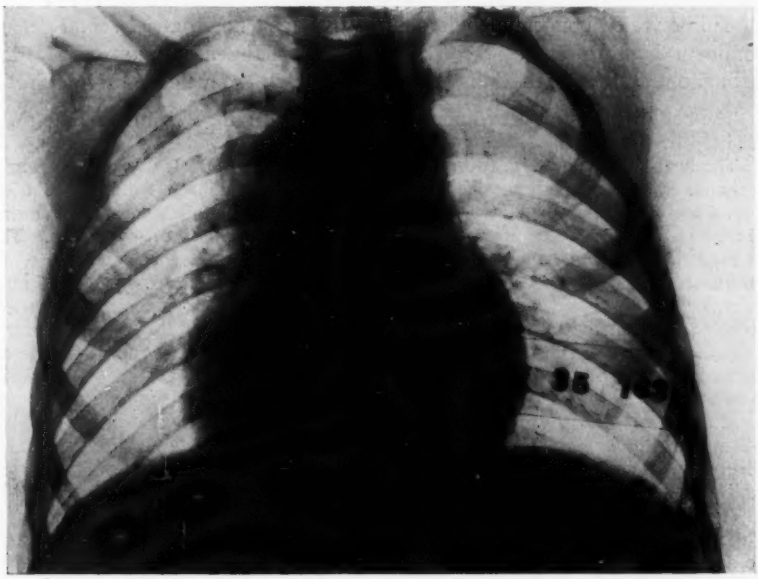


FIG. 3.—Teleradiogram of heart: note the central position, the prominence of the ascending aorta, the shadow external to the ascending aorta (probably the superior vena cava), and the rounded, bulky left ventricle.

The diagnosis was congenital heart disease with permanent cyanosis, clubbing, and polycythæmia, but the exact lesion or combination of lesions could not be determined. For while the clinical features suggested marked pulmonary stenosis or atresia, the X-ray and cardiographic evidence left little doubt that the left side of the heart was carrying the burden.

The subsequent course threw no further light on the matter. She was observed for nearly two years, and repeated clinical, radioscopic, and cardiographic examinations merely confirmed the above findings. On one occasion

she was admitted with athetoid movements of the right arm, when her blood count was as high as 11,200,000 red cells per c.mm., and the hæmoglobin 124 per cent. At that time it was shown that the viscera were not transposed. On another occasion, night sweats, general malaise, slight fever, a palpable spleen, and a few granular casts and red cells in the urine, made me suspect superimposed bacterial endocarditis. The blood urea was 30 mg. per 100 c.c., the specific gravity of the urine varied between 1005 and 1025, and there was no albuminuria. A blood culture was not obtained.

On March 5, 1937, she was readmitted with frequent syncopal attacks associated with deepened cyanosis, and on March 29 she died in one of them. These attacks were similar to those of which she had complained for many years, and which have already been described. At this time she was eight years old.

Post-Mortem Examination

This revealed congenital pulmonary valvular stenosis, atrial septal defect, dominant hypertrophy of the left ventricle, and intense venous congestion throughout all the viscera, but especially marked in the meninges and brain.

The body was that of a well nourished female child: the weight was 36 lb.; the height, 4 ft.

The heart weighed 182 g. (normal for this age 95–105 g.). It was almost square in shape. The aorta was prominent, and the dominant chamber was the left ventricle (Fig. 4 and 5). This was dilated and considerably hypertrophied, its wall measuring 10 mm. in thickness at the apex, 16 mm. at the centre, and 18 mm. at the base. The interventricular septum bulged to the right, and was intact. The right ventricle was contracted, and its wall measured 12 mm. at the site of the moderator band, 8·5 mm. on either side of it, and 6 mm. at the base. The outflow tract of the right ventricle was elongated and narrow. The pulmonary valve (Fig. 4) consisted of two cusps, mainly fused together, leaving a narrow aperture slightly less than 5 mm. in diameter. Around the free margin of the cusps was a ring of granular, wart-like, vegetations, composed microscopically of hyaline thrombi without cellular reaction. The left auricle was considerably dilated and somewhat hypertrophied, its wall measuring 1–2 mm. in thickness. The right auricle was also enlarged, but a little less conspicuously. There was a large defect of the atrial septum, at the site of the fossa ovalis, the aperture measuring 2 cm. in diameter (Fig. 5).

The aortic, mitral, and tricuspid valves were normal. There was no coarctation of the aorta; the great vessels were normal; the coronary arteries were normal; and the ductus arteriosus was closed.

Microscopically, the muscle fibres of the left ventricle were thicker than those from any other chamber. Apart from some increased cellularity of the interstitium, and a trace of perivascular fibrosis here and there, there were no significant microscopic changes.

The other autopsy findings simply showed the effects of intense venous congestion, all the viscera being involved. The bone marrow was very hyperplastic, and microscopically this was shown to be due to normoblastic proliferation. The meninges over the whole of both hemispheres were intensely vascular and deeply congested, the vessels lying so close together as to be almost touching. The vascularity and congestion were such as to form a deep purple membrane, through which the underlying brain could not be seen. The grey matter of the brain, over practically the whole of both hemispheres, showed a fine granularity, and one section was deeply congested. On the posterior lateral aspect of the left occipital lobe there was an

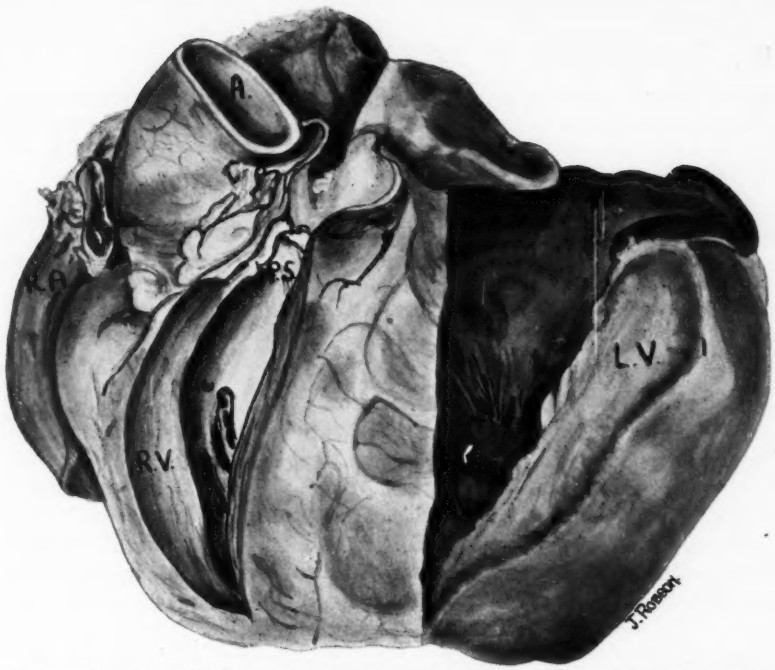


FIG. 4.—Drawing of the heart (to scale) from the front: A., aorta; L.V., left ventricle; P.S., pulmonary stenosis; R.V., right ventricle; R.A., right auricle.

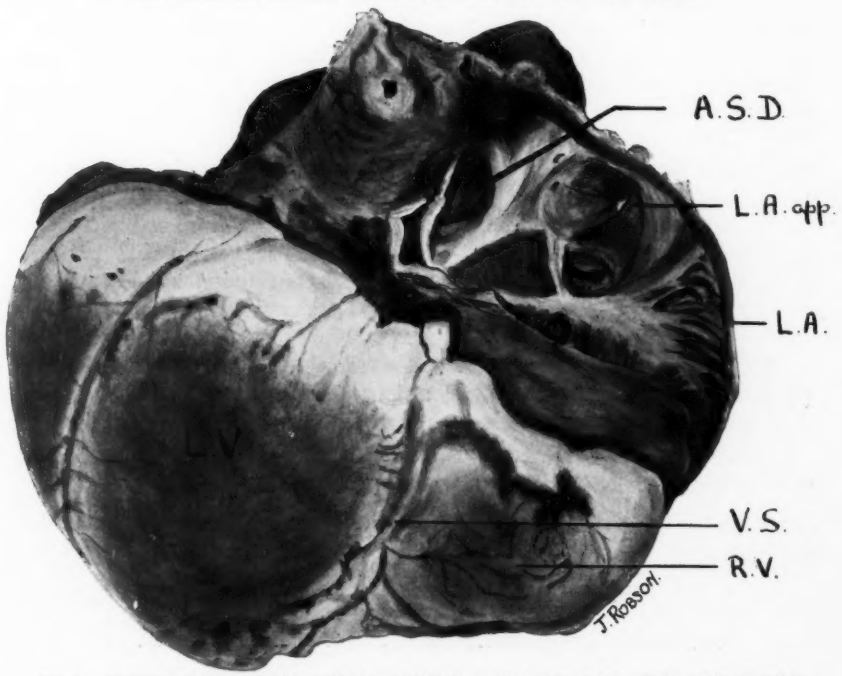


FIG. 5.—Drawing of the heart (to scale) from behind: L.V., left ventricle; R.V., right ventricle; L.A., left auricle; L.A. app., left auricular appendage; A.S.D., atrial septal defect at site of fossa ovalis.

area of softening, limited to the grey matter, which in this area was practically absent, being represented by a little semi-fluid matter. The pons, medulla, and cerebellum were less affected than the rest of the brain. The white matter appeared normal throughout.

Dr. Greenfield kindly examined the sections of the brain and meninges, and concluded that all the changes could be attributed to intense congestion. Some of the nerve cells were indistinct and shrunken. The nuclear chromatin had lost its normal pattern, and stained darkly and diffusely. The nucleolus was not, as a rule, visible. In some areas there was complete disappearance of all nerve cells. There was no apparent gliosis or other change. In the occipital lobe, the softened area previously described, consisted of collapsed neuroglial reticulum devoid of nerve cells.

Discussion

It is clear that this was a case of pulmonary valvular stenosis complicated by wide patency of the interauricular septum, there being no other abnormalities. Uncomplicated atrial septal defect is held to be responsible for the gross enlargement of the chambers on the right side of the heart which is associated with it (Bedford, Papp, and Parkinson, 1941). In these cases it is believed that there is a considerable shunt of blood from the left to the right auricle, there being reason to think that the pressure in the left auricle is normally higher than that in the right—for consider the mechanism of closure of the foramen ovale, and the behaviour of paradoxical emboli. The extra work thus given to the right side of the heart is the simplest explanation of the great enlargement found. The sudden development of extreme cyanosis when the right ventricle begins to fail is readily understood on the supposition that the rising pressure in the right auricle effects a reversal of flow through the atrial septal defect. In the case recorded here, extreme cyanosis was lifelong, and the left side of the heart was most enlarged, facts which indicate that the shunt was perpetually from right to left. It follows that the pressure in the right auricle must have been higher than that in the left. It is not obvious why this should have been so, for right ventricular failure was not clinically apparent. Had tricuspid stenosis been present as a congenital defect the case would have presented no difficulties, but the tricuspid valve was normal. One is driven to the conclusion that the pulmonary stenosis was responsible for the raised pressure in the right auricle.

Summary

A case is described which presented the following features:
(1) pulmonary valvular stenosis, (2) atrial septal defect, (3) left ventricular dominance, and (4) extreme permanent cyanosis.

The question arises whether this will prove a clinically recognizable congenital syndrome, or whether this is a freak case.

My thanks are due to the chief medical officer of the London County Council for his permission to publish this case; and I wish to express my appreciation and gratitude to Mr. J. Robson for his careful drawings of the heart.

Reference

D. E. Bedford, C. Papp, and J. Parkinson. *Brit. Heart J.* (1941), 3, 37.

INFARCTION OF THE CARDIAC AURICLES (ATRIA): CLINICAL, PATHOLOGICAL, AND EXPERIMENTAL STUDIES

BY

E. H. CUSHING, H. S. FEIL, E. J. STANTON, AND W. B. WARTMAN

From the Departments of Medicine and Surgery and the Institute of Pathology of Western Reserve University and the University Hospitals of Cleveland, U.S.A.

Received December 8, 1941

Infarction of the auricles * (atria) of the heart is generally considered a rare lesion of little clinical importance and few cases are reported. However, a study of the records at the University Hospitals of Cleveland showed that the auricles were involved in 31 (17·0 per cent) of 182 cases of myocardial infarction that were proven at autopsy during the seven year period from 1934 to 1940. This is the highest incidence of auricular infarction in any reported series, but is thought to be near the true incidence of the lesion, because during the period of investigation the auricles were specifically examined for infarcts.

In a previous publication (Feil, Cushing, and Hardesty, 1938) two cases of infarction of the right auricle were encountered in a pathological study of 34 cases of acute myocardial infarction. Bean (1938) reported a series of 300 cases of myocardial infarction in which two instances of auricular infarcts were encountered at autopsy. Auricular fibrillation was present in one of these cases. Clowe, Kellert, and Gorham (1934) described a verified case of rupture of the right auricle. They analysed 54 proved cases that had been reported and found that the rupture had occurred in the right auricle in 70 per cent of them. Laignal-Lavastine, Liber, and Bidou (1934) found a ruptured hæmorrhagic infarct in the right auricle, following thrombosis of the first portion of the right coronary artery and its auricular branches. Clerc and Levy (1925) studied a patient with mitral stenosis and infarction of the external and superior portions of the right auricle. The electrocardiogram showed what they termed " fibrillo-flutter." Lisa and Ring (1931) described rupture of the left auricle in a heart in which there was advanced coronary arteriosclerosis and a recent thrombus in the anterior descending branch of the left coronary artery. Daven-

* In the anatomical and pathological portions of this paper, starting with the section headed *Pathological Observations* on p. 20, a distinction is made between the words " atrium " and " auricle " in accordance with the B.N.A. terminology. Atrium is defined as the entire chamber, either left or right, at the base of the heart above the ventricle. The word " auricle " is reserved for the ear-like appendage of the atrium. In the discussion of the physiological and clinical aspects of the lesion, deference is made to long and common usage so that when the adjective " auricular " is employed it refers to the entire chamber and not to the appendage. Thus such terms as " auricular fibrillation," " auricular flutter " and " auricular mechanism " do not imply localization to the appendage.

port (1928) reported 92 cases of proved cardiac rupture in which the right auricle was affected in 3 per cent and the left in 1 per cent. Krumbhaar and Crowell (1925) studied 22 cases of rupture of the heart and reviewed 632 reported cases. The right auricle was involved in 5 per cent and the left in 2 per cent, and infarction was mentioned as a frequent cause. Infarction of the auricular musculature was seen three times in von Glahn's (1936) pathological material.

Recently Langendorf (1939) studied a case of auricular infarction, which was verified by autopsy, and reported deviations from the isoelectric line of the P–Q segment in the second and third leads of the electrocardiogram. In experimental injury of the atria, changes have been described in the P wave and in the P–Q level by Hahn and Langendorf (1939). Abramson, Fenichel, and Strookhoff (1938) reported elevation of the P–Q interval (lead I) in dogs, following necrosis of the left atrium experimentally produced, and depression of the interval after necrosis developed in the right atrium. They suggested that the deviation of the P–Q interval in man is due to necrosis of the auricle and is analogous to the deviation of the S–T interval which occurs in ventricular ischaemia. In experiments in which the atrial arteries of rabbits were clamped, Lambert (1937) found changes in the auricular complex of the electrocardiogram. The most interesting phenomena were the depression of the P–Q segment, which he termed "cupule," and occasionally a diphasic or inverted P wave. Conderelli (1929), on the other hand, was unable to produce electrocardiographic changes after temporary occlusion of the right atrial vessels. Sanders (1939) injected 95 per cent alcohol into the atria of dogs and found that nodal rhythm, wandering pacemaker, auricular extrasystoles, and paroxysmal auricular tachycardia developed when the necrosis involved the sinus node. Elevation of the auricular S–T segment in lead I with an upward bowing and an auricular Q wave frequently followed injection of the left auricle. Necrosis of the right auricle caused similar auricular S–T changes in leads II and III. Changes in the P wave consisted of broadening and inversion, diminution or increase in amplitude, and slurring and notching. Sometimes a Q or S wave was seen, or M or W complexes.

The present study was undertaken to determine: (1) the clinical and electrocardiographic criteria that are diagnostic of auricular (atrial) infarction; (2) the incidence of auricular involvement in autopsied cases of myocardial infarction; and (3) the electrocardiographic changes in experimental auricular (atrial) infarction.

Clinical Observations in Human Auricular Infarction

Thirty-one cases of auricular (atrial) infarction were found in 182 cases of myocardial infarction (17 per cent), culled from 2704 consecutive autopsies. Electrocardiograms were taken in 23 cases and the findings are given in Table I. Abnormal auricular mechanism was present in 17 of the 23 cases (74 per cent). On the other hand, in 91 verified cases of ventricular infarction without demonstrable auricular infarction, in which electrocardiograms had been taken,

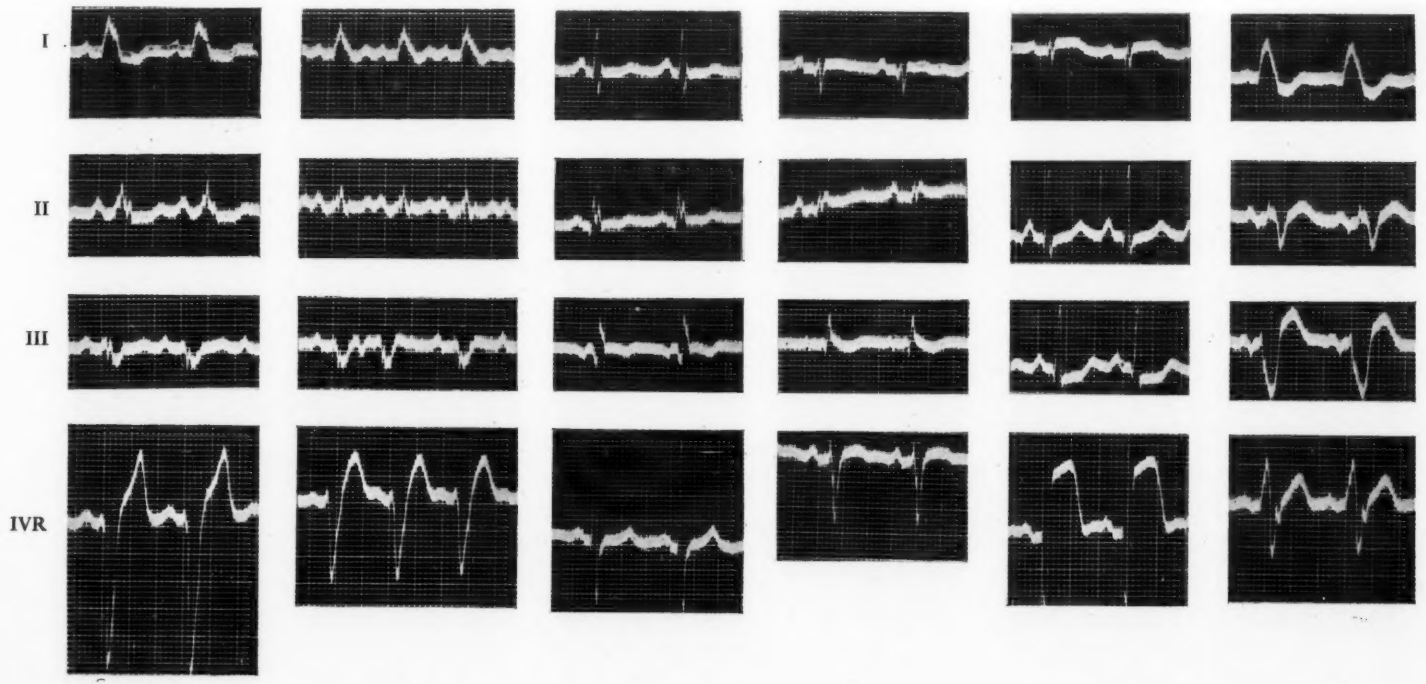


FIG. 1.—(A) Case 12. 20–4–38. Before infarction: left bundle branch block. (B) Case 12. 25–7–39. After infarction: depression of P–Q in leads II and III and bundle branch block.
(C) Case 29. 11–8–37. Before infarction. (D) Case 29. 7–10–37. After infarction: depression of P–Q in leads I, II, and IVR, and RS–T changes in IVR, typical of anterior and apical infarction.
(E) Case 25. After infarction: depression of P–Q; QRS–T changes typical of anterior and apical infarction.
(F) Case 31. After infarction: depression of P–Q in leads II and III and left bundle branch block.

there was evidence of disturbed auricular rhythm in only 8 cases (8 per cent). In the patients with infarcts of the auricles, auricular fibrillation was observed in 9 cases, auricular premature beats in 4, auricular flutter in 2, and sinus arrest and wandering pacemaker in 1 case each. The depression of the P–Q level (Fig. 1) described by Lambert and Langendorf occurred in 5 instances (22 per cent), but the value of this sign is doubtful because of its occurrence in normal people (Shipley and Hallaran, 1936). In the two cases with infarction of the sinus node, sinus arrest was present in one and auricular fibrillation in the other.

Pathological Observations in Human Atrial Infarction

A summary of the pathological observations in the cases of atrial * infarction is given in Table I. Before discussing these findings, it should be emphasized that many cases will be overlooked at autopsy, unless the existence of the lesion is kept constantly in mind. The gross recognition of atrial infarcts may be difficult and in questionable cases many blocks must be taken for microscopic examination. In this study, routine blocks were taken from the region of the sinus node, from both auricles, and from the lateral wall. Whenever mural thrombi were found in the atria, a suspicion of infarction was at once entertained and appropriate blocks were taken through such areas.

Fig. 2 (on p. 25) shows the location of the infarcts in the atria. The great majority of them were in the right atrium (27 times) in contrast to the occasional involvement of the left atrium (5 times). Most of the infarcts occurred in the auricles while the region of the sinus node was affected in only two instances. Multiple infarcts were found in two hearts.

Although the atrial arteries were carefully inspected after fixation by means of closely placed transections, gross occlusion was demonstrated in only one case. No doubt obstruction would have been found more frequently, had the coronary arteries been injected.

On the other hand, the anterior descending and circumflex branches of the left coronary artery, as well as the main right coronary artery were often diseased and serious stenosis or occlusion was encountered in 23 of the 31 cases. Either recent or remote infarcts of the ventricular myocardium were present in 22 of the cases. These were all in the left ventricle and interventricular septum, and often were multiple. Two other cases showed acute diffuse myocarditis and one chronic myocarditis. This left 6 hearts (*Cases* 3, 16, 17, 21, 26, and 28) in which auricular infarcts were the only lesions other than hypertrophy. With the exception of *Case* 26 all the infarcts were recent. Electrocardiograms had been taken in 4 of the 6 patients (*Cases* 3, 16, 21, and 26) and 2 had auricular fibrillation, 1 auricular flutter, and 1 low voltage of QRS. *Case* 3 died of heart failure; *Case* 16 suffered from malignant hypertension and died of uræmia; *Case* 17 had hypertension with cardiac decompensation, as well as acute pyelonephritis, and died of broncho-pneumonia; *Cases* 26 and 28 had hypertension and died of cardiac failure. It is, of course, unlikely that the auricular infarcts

* See footnote on p. 17.

ABBREVIATIONS USED IN TABLE I

A.F.	=	Auricular fibrillation	L.C.	=	Circumflex ramus left coronary artery
A.Fl.	=	Auricular flutter	L.D.	=	Descending ramus left coronary artery
ant.	=	Anterior	N	=	Normal findings
A.P.B.	=	Auricular premature beat	post.	=	Posterior
Art.	=	Arteriosclerosis	R.A.	=	Right auricle (atrium)
dep.	=	Depressed	R.A.A.	=	Right auricular appendage (auricle)
I.V.S.	=	Interventricular septum	R.S.R.	=	Regular sinus rhythm
L.A.	=	Left auricle (atrium)	R.V.	=	Right ventricle
L.A.A.	=	Left auricular appendage (auricle)	Sten.	=	Stenosis
lat.	=	Lateral	Vent. Tachy.	=	Ventricular tachycardia
L.B.B.B.	=	Left bundle branch block			

TABLE I

SUMMARY OF CLINICAL AND PATHOLOGICAL FINDINGS IN HUMAN INFARCTION OF THE CARDIAC ATRIA

Case No.	Age in Years	Sex	Colour	Electrocardiogram	Heart Weight in Grams	Location and Age of Atrial Infarct	Mural Thrombi in Atria	Lesions in Main Coronary Arteries	Location and Age of Ventricular Infarcts
1	80	F	W	N	520	L.A., post; recent	N	L.D.; remote L.C.; remote R.C.; recent Art. +++Sten.	L.V., post basal; remote. Acute myocarditis
2	54	F	W	A.F.	400	R.A.A.; recent	R.A.A.; recent L.A.A.; recent	Art. ++	L.V. and I.V.S., ant. and apical; remote
3	63	F	W	A.F.; remote ant. infarct.	450	R.A.A.; recent	R.A.A.; recent	Art. +++Sten.	N
4	74	M	W	A.F.; remote ant. infarct.	640	R.A.A.; recent and remote	R.A.A.; recent L.A.A.; remote	L.D.; recent and remote L.C.; remote R.C.; remote in A. branch Art. +++	L.V. and I.V.S.: 1. recent and remote at apex 2. recent post. and basal

TABLE I—*continued*

Case No.	Age in Years	Sex	Colour	Electrocardiogram	Heart Weight in Grams	Location and Age of Atrial Infarct	Mural Thrombi in Atria	Lesions in Main Coronary Arteries	Location and Age of Ventricular Infarcts
5	70	M	W	A.F.; recent post. infarct.	700	R.A.A. and R.A., post.; recent	R.A.; recent	L.C.; recent R.C.; recent Art. +++Sten.	L.V., lat. and post. basal I.V.S., post. R.V., ant. at apex; all recent
6	52	M	W	N	570	R.A.A.; recent	R.A.A.; recent	Art. +	L.V.; acute myocarditis
7	45	M	B	A.F.	650	R.A.A.; recent and remote	R.A.A.; recent	Art. +	L.V., apex; recent
8	64	M	W	A.F.	560	R.A., lat.; recent	R.A., lat.; recent	Art. +++Sten.	L.V., ant. basal; recent L.V., ant. apical; remote
9	69	F	W	R.S.R , sinus arrest; recent ant. infarct.	480	R A., post.; near sinus node	N	Art. +++Sten.	L.V. and I.V.S., apex; recent
10	86	M	B	N	550	R.A.A.; recent	R.A.A.; recent	Art. ++	Acute myocarditis
11	55	M	W	A.P.B. R.S.R.; recent post. infarct.	800	R.A.A.; remote	N	L.D.; remote R.C.; recent Art. +++Sten.	L.V., apex.; remote L.V., post.; recent R.V., post.; recent I.V.S., post.; recent
12	59	M	W	A.P.B. R.S.R. L.B.B.B ; $P–Q_{2,3}$ dep	600	R.A.A.; recent and remote	R.A.A.; recent	Art. +	Chronic myocarditis Healed mural endocarditis

13	64	M	W	N	260	R A A.; recent	R.A.A.; recent	R.C.; recent Art. +++Sten.	L.V., apex; recent and remote L.V. and R.V., post. basal; remote
14	58	F	W	A.F.	580	L.A.A.; remote	R.A.A.; recent L.A.A.; recent	Art. +	L.V., lat. and basal; remote
15	57	M	W	A.P.B.; low voltage QRS; remote ant. infarct.	630	L.A.A.; remote	N	L.D.; remote Art. +++Sten.	L.V., R.V., and I.V.S., apex; remote
16	45	F	B	A. Fl.	690	R.A.A.; recent and remote	R.A.A.; recent	Art. +++	N
17	77	M	W	N	790	R.A.A.; recent	R.A.A.; recent	Art. +	N
18	49	M	W	A.F.; T_1 neg.	640	R.A., lat.; recent and remote	R.A.; recent	L.D.; remote Art. +++Sten.	L.V. and I.V.S., apex; remote
19	50	M	W	N	350	R.A.A.; remote	N	L.D.; remote Art. ++Sten.	L.V., apex; remote
20	38	M	B	R.S.R.; P–Q_4 dep.	650	R.A.A.; recent and remote	R.A.A.; remote	L.C.; recent Art. +++Sten.	L.V., post. basal; recent
21	35	F	B	R.S.R.; low voltage QRS	200	R.A.A.; remote	N	Art. ++Sten.	N
22	58	M	W	A.P.B. R.S.R.; remote ant. and post. infarct.	520	R.A.A.; recent	R.A.A.; recent	L.D.; recent Art. +++Sten.	L.V., lat.; recent L.V., post. basal; remote
23	76	M	W	R.S.R.	470	R.A., post.; remote	N	L.D.; recent and remote	L.V., post. basal; recent and remote

TABLE I—*continued*

Case No.	Age in Years	Sex	Colour	Electrocardiogram	Heart Weight in Grams	Location and Age of Atrial Infarct	Mural Thrombi in Atria	Lesions in Main Coronary Arteries	Location and Age of Ventricular Infarcts
24	74	M	W	N	650	R.A.A.; remote	R.A.A.; recent	R.C.; remote Art. +++Sten.	L.V., apex; remote
25	55	M	W	R.S.R.; P–Q_4 dep.	580	R.A.A.; recent	R.A.A.; recent	L.D.; recent Art. +++Sten.	L.V. and I.V.S., apex; recent
26	47	M	W	A.F.; remote ant. infarct.	600	R.A.A.; remote R.A. and L.A.; acute and chronic myocarditis and endocarditis.	R.A.A.andL.A.A.; recent	L.C.; remote Art. ++	N
27	56	M	W	A.F., vent. tachy.; recent post. infarct.	350	L.A.A.; remote Multiple small infarcts	L.A.A.; recent	L.C.; recent R.C.; recent and remote	L.V. and I.V.S., lat. and post.; recent
28	60	M	W	N	500	R.A.A.; recent	R.A.A.; recent	N	N
29	50	M	W	R.S.R., wandering pacemaker; P–Q_1 dep. recent ant. infarct.	650	R.A.A.; recent and remote	R.A.A.; recent	L.D.; recent L.C.; recent Art. +++	L.V. and I.V.S., apex, lat. and post.; recent
30	57	M	W	N	450	R.A., lat.; remote L.A., lat.; remote	R.A.A.; remote	R.C.; remote Art. +++Sten.	L.V. and I.V.S., apex; remote
31	67	F	W	R.S.R. L.B.B.B.; P–$Q_{2,3}$ dep.	520	R.A.A. and R.A., lat.	R.A.A.; recent	Art. +++Sten.	L.V., post. lat.; recent

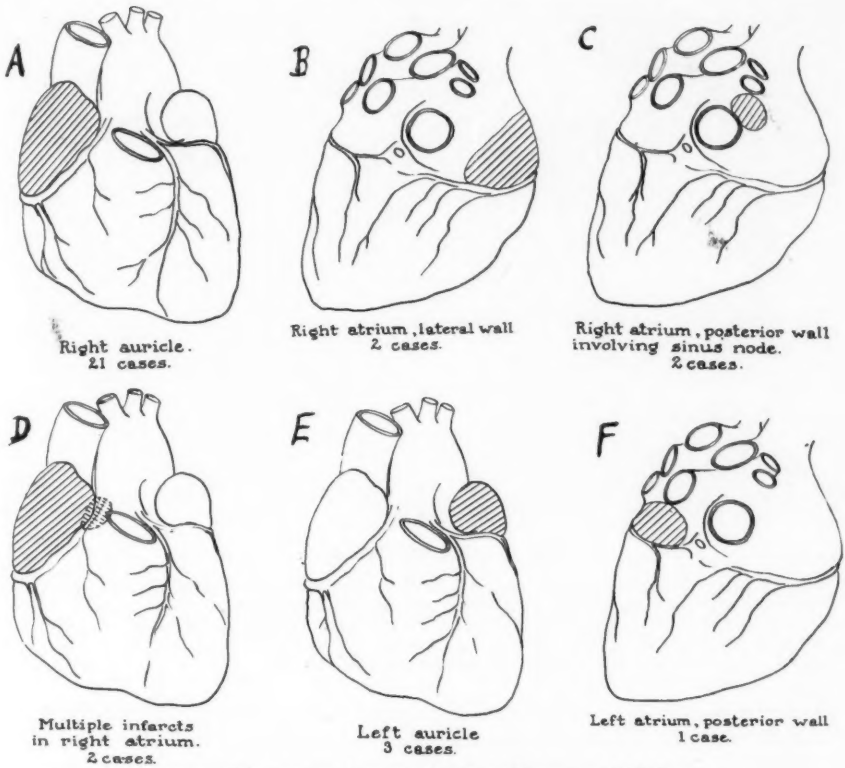


FIG. 2.—Diagrams to show the situation of human atrial infarcts.

(A) Right auricle, 21 cases. (B) Right atrium, lateral wall, 2 cases. (C) Right atrium, posterior wall involving sinus node, 2 cases. (D) Multiple infarcts in right atrium, 2 cases. (E) Left auricle, 3 cases. (F) Left atrium, posterior wall, 1 case.

caused the death of these patients, but it is conceivable that they were a contributory factor.

The gross appearance of atrial infarcts was similar to that commonly seen in the ventricles (Fig. 3). It is worthy of note that in 26 cases, mural thrombi were found tightly adherent to the endocardium over the infarcted area.

Microscopically all stages of infarction were observed. The earliest changes consisted of hyperæmia and hæmorrhage with swelling of the muscle fibres and necrosis and exudation of leucocytes, followed later by hæmosiderin pigmentation and scarring. Often the muscle bundles in adjacent areas were swollen with vacuolated cytoplasm and large pleomorphic and hyperchromatic nuclei. In most cases the infarcts were massive, but occasionally small multiple lesions, separated from each other by intact myocardium, were observed. Massive infarction was usually accompanied by mural thrombosis of the endocardium, and when organization had occurred it was difficult to distinguish

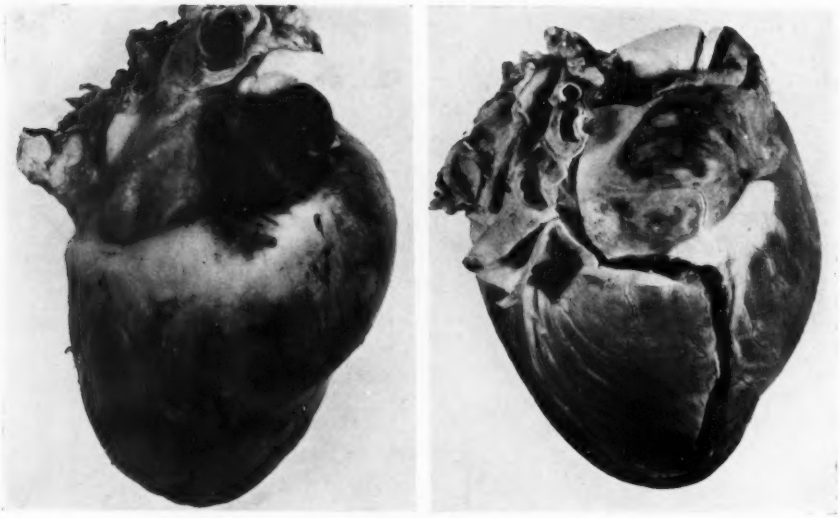


FIG. 3.—Atrial infarcts in dogs.

(A) Recent infarction in right auricle following experimental ligation of the main right coronary artery. A large infarct of the right ventricle was also present.

(B) Naturally occurring infarct in right auricle of a dog. No ventricular infarction. The localization of these lesions in the auricles, the dusky purple discoloration, and the presence of mural thrombi are also characteristic of the human lesions.

between necrotic myocardium and the thrombus. In such cases the Weigert stain was helpful, for by demonstrating the elastic lamina of the endocardium, the line of demarcation between muscle and thrombus could be clearly seen. When the infarct was not accompanied by mural thrombosis, there was nearly always a narrow border of surviving myocardium immediately beneath the endocardium. Microscopic study revealed small thrombotic arteries in several instances.

DISTRIBUTION OF THE CORONARY ARTERIES OF THE ATRIA IN DOGS AND IN MAN

In this study of human atrial infarction the electrocardiographic changes described by Lambert and by Langendorf were not seen frequently. In view of this discrepancy, infarction of the atria of the heart was produced experimentally in dogs. Before describing the findings, it is necessary to review the distribution of the coronary arteries of the atria in dogs since it is different from that in man.

The atrial vessels of the dog's heart (Fig. 4) have a fairly constant distribution (Meek, Keenan, and Theisen, 1928–29). One or two vessels arise from the right coronary artery, shortly after its origin, and supply the surface of the right auricle and the superior vena cava. The lateral branch supplies the posterior

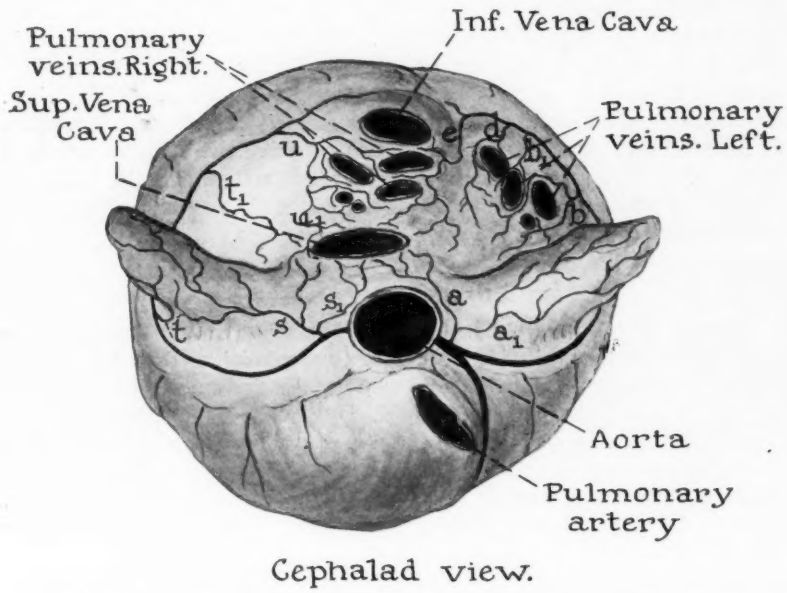


FIG. 4.—Distribution of the atrial branches of the coronary arteries in the dog.

a	Left anterior	*s*	Right anterior
a_1	Left anterior accessory	s_1	Right anterior accessory
b	Left intermediate	*t*	Right intermediate
b_1	Left intermediate accessory	t_1	Right intermediate accessory
d	Left anterior pulmonary branches	*u*	Right posterior
e	Left posterior	u_1	Sulcus artery

aspect of the right atrium, and there is a constant posterior branch that supplies the tissue about the pulmonary veins and anastomoses with the anterior branch about the superior vena cava. From the left coronary artery two branches arise close to the origin, one supplying the auricle and the other going upwards about the aorta to the vena cava. Posteriorly on the left side there is a lateral branch which ramifies about the pulmonary veins and goes upward toward the superior vena cava.

In man there are usually two branches from the right coronary artery (Gross, 1921). The first arises near its origin and comes behind the aorta, 5 mm. above the auriculo-aortic groove. It supplies the anterior wall of the right atrium and sends branches to the auricle. It then passes through the septum to reach the posterior wall, where it surrounds in ring fashion the superior vena cava. This branch may arise from the left coronary artery near its origin; it encircles the base of the left auricle, ascending the external surface of the left atrium to the region of the superior vena cava, where it terminates as an arborization about the superior vena cava or passes immediately after its origin along the anterior wall of the left atrium to within 5 mm. of the auriculo-aortic groove. On reaching the inter-atrial septum, it

turns up and crosses on the superior aspect of the right atrium between the superior vena cava and the right auricle. The left coronary artery shows, in addition to the above branches, one or two smaller atrial branches, which distribute themselves over the anterior and superior surfaces of the left atrium. Occasionally, a left lateral atrial branch extends around the posterior surface of the atrium to reach the opening of the superior vena cava. The second branch of the right coronary artery is less constant and supplies the aortic face of the right auricle and the superior surface of the right atrium. There are a few small and inconstant atrial vessels from both sides.

Experimental Infarction of the Atria

All experiments were performed under aseptic conditions upon medium-sized dogs, anæsthetized with ether which was administered by intermittent positive pressure insufflation. Pre-operative medication consisted of morphine sulphate, 0·01 g., given hypodermically one hour before the experiment.

The heart was exposed and supported in a cradle constructed by suturing the widely opened pericardium to the chest wall. Dissection of the right or the left circumflex coronary artery was begun at its origin and continued distally, each atrial branch being exposed, temporarily occluded several times, doubly ligated with fine silk, and finally cut.

Electrocardiograms were taken, employing the standard three leads and the chest lead IVR as follows: (1) a normal control before the start of the experiment; (2) one hour after the administration of morphine; (3) after ether anæsthesia; (4) after opening the chest; (5) after opening the pericardium ; (6) after construction of the pericardial cradle; (7) before and after each temporary occlusion of the individual atrial arteries; (8) before and after permanent ligation and section of each atrial artery; (9) at the conclusion of the experiment; and (10) daily thereafter. Chest leads were not taken after opening the thorax.

One or more atrial arteries were ligated in 18 dogs. Multiple operations at 12 to 21 day intervals on the same dog were performed and all right atrial branches were ligated in 11, all left atrial branches in 7, and the arteries of both atria in 4 instances.

In four dogs the effect of chemically produced necrosis of the atrial myocardium was studied. In one animal various portions of the right atrium were painted with liquid phenol, and in three dogs the actual cautery was applied successively to the right and left atrium.

After sacrifice the hearts were injected through the right and left coronary arteries with barium sulphate gelatin after the method of Louis Gross (1921), chilled, X-rayed, and placed in 10 per cent formalin. Gross and microscopic examination of the hearts was made following the usual routine.

Ligation of Right Atrial Arteries

Dog 38–10. Artery s_1 ligated. Immediately afterwards P_2 and P_3 showed increased amplitude and returned to normal two hours later. There were no P–Q changes.

Daily electrocardiograms for twenty-two days showed no abnormality. Twenty-three days later, arteries t and t_1 were ligated. After operation P_2 was slightly increased without P–Q changes, and one day later the record was entirely normal. Twenty-one days later the animal was sacrificed and an organizing infarct of the right auricle and the atrium, as far as the acute margin of the heart, was found.

Dog 38–12. Arteries s_1 and s ligated. The electrocardiogram showed slight elevation of P_2 and P_3 which persisted for twenty-four hours. Nineteen days later, pathological studies showed a small organizing infarct of the right auricle.

Dog 38–13. Artery s_1 ligated. No electrocardiographic change. Twenty-one days later, artery t_1 was ligated. The electrocardiogram remained normal.

Dog 38–14. Artery s_1 ligated. No electrocardiographic change.

Dog 39–273. Artery t (small vessel) ligated. Wandering pacemaker developed and persisted about fifteen minutes. Following this, artery u was ligated and the electrocardiogram showed slight changes in contour of P_1. Artery t_1 was ligated with no further change in the electrocardiogram. Artery s was then ligated and auricular tachycardia developed. P_2 and P_3 were broad and notched with increased amplitude. Forty-eight hours later the electrocardiogram was normal. After sacrifice the injected specimen showed that all right branches had been successfully ligated. Injection of the left coronary artery showed the barium to pass from the left atrial branches and fill the right atrial arteries. There was a recent infarct of the right auricle.

Dog 39–278. All right atrial arteries were ligated; following this slight but definite depression of P–Q developed, and disappeared within twenty-four hours. Fifteen days later the dog was sacrificed, and injection showed that all arteries to the right atrium had been ligated. Excellent interatrial anastomoses with the left atrial artery were present. Microscopic studies showed infarction of the right auricle with beginning organization.

Dog 39–280. All right atrial arteries were ligated successively and nodal rhythm resulted. The dog died twelve hours after the experiment. Injection of the coronary arteries showed that all branches to the right atrium were successfully ligated. Microscopic sections were not obtained.

Dog 39–282. All right atrial arteries were ligated. The electrocardiogram remained normal throughout. Injection of the coronary arteries with barium showed all right atrial arteries to have been ligated. Microscopic studies showed a recent infarct of the right auricle.

Ligation of Left Atrial Arteries

Dog 39–258. Artery a ligated and cut. No electrocardiographic change. The dog died of ventricular fibrillation during the experiment. Death was attributed to insufficient lung inflation.

Dog 39–260. Artery a ligated. P_1 became isoelectric, P_2 and P_3 were inverted, and the P–Q interval remained normal. These changes persisted throughout the experiment. Artery b_1 was then ligated without further change. On the first day after operation, nodal rhythm appeared and persisted four days. From the fifth to the ninth days, wandering pacemaker was present. On the tenth day the rhythm became normal and remained so. At autopsy, recent infarction of the lateral wall of the left atrium was present.

Dog 39–259. Arteries b_1 and b ligated. There were no electrocardiographic changes. Death occurred during the experiment as the result of pressure on the heart.

Ligation of Right and Left Atrial Arteries

The arteries of both atria were successfully ligated in four dogs. In one dog this was done in a one-stage operation and in three dogs by multiple stages.

Dog 39–280. Ligation of arteries *u* and *t* produced no electrocardiographic changes. Following ligation of arteries *s* and s_1 nodal rhythm resulted. Ligation of all left atrial arteries produced no further changes.

Dog 39–264. The first operation was performed on September 21, 1939, when artery *a* was ligated, after which P_1, P_2, and P_3 became isoelectric and triphasic with wandering pacemaker. Artery *b* was then tied without significant change. After operation, wandering pacemaker and slight variation in size and shape of P_1, P_2, and P_3 were present.

On October 2, artery *u* was ligated without change in the electrocardiogram. A small artery proximal to *u* was ligated and slight variation in contour of the P wave appeared. Ligation of artery *t* produced no further change. After artery *s* was ligated, P_3 became diphasic with slight terminal depression. Ligation of artery s_1 produced no further change. After closure, 1:1 flutter developed (rate 279). On the first day after operation the rate was 220 with premature auricular beats. On the second and third days the rate was 226 and 284 respectively. On the fourth and fifth days A-V block, nodal extrasystoles, slight P–Q depression, and prolonged P–R interval (0·17) were present. On the following three days auricular flutter was present (auricular rate, 374; ventricular rate, 192). On the tenth day the rhythm was normal and remained so. At autopsy there were organizing infarcts in the lateral walls and auricles of both atria.

Dog 39–276. At the first operation on September 27, 1939, artery *s* was ligated and the electrocardiogram remained normal. All remaining atrial arteries were then successively ligated without electrocardiographic change.

At the second operation on October 3, arteries *a*, *b*, and b_1 were ligated. No electrocardiographic change occurred either during the experiment or after the operation. Twenty days later the animal was sacrificed and all the left and right atrial arteries were found to have been ligated. There were organizing infarcts in the right and left auricles.

Dog 39–267. The first operation was performed on September 25, 1939, and arteries *t* and *u* were ligated. The $P–Q_2$ showed a depression of 1 mm., which continued for one day.

The second operation was performed on October 9 at which time arteries *a*, *b*, and b_1 were ligated. Only slight transient changes occurred in the contour of the P wave, and these did not persist after the operation.

The third operation was performed on October 17 when arteries *s* and s_1 were ligated. This was followed immediately by a low amplitude P wave which, however, returned to normal immediately after operation. On October 19 wandering pacemaker developed and disappeared within 24 hours, only to reappear on October 25.

Post-mortem organizing infarcts involving the lateral walls and auricles of both atria were found.

All the hearts were carefully examined for other evidence of disease, but none was found. No enlargement occurred. The main coronary arteries were widely patent and followed a normal distribution. No infarcts were found in either right or left ventricles or the interventricular septum and there was no myocarditis, endocarditis, or valvulitis. Numerous dense adhesions often containing foci of heterotopic bone were present in the atrio-ventricular sulcus at the operative site.

In all the experiments described so far, the main right and left coronary arteries were normal, which, of course, was very different from the human cases where the main coronary arteries were usually seriously diseased and showed marked stenosis. The human lesions were usually massive, were accompanied by mural thrombosis, and involved the endocardium. In the dogs, on

the other hand, no mural thrombi were formed, the infarcts consisted of small multiple scars or foci of fresh necrosis separated by bands of healthy muscle, and there was always a narrow zone of healthy myocardium immediately beneath the endocardium. Because of these differences between the experiments in the dog and the spontaneous human lesions, ligation of the main right coronary artery close to its origin was carried out in three dogs.

Dog 41–290. Shortly after ligation, S–T_1 became depressed and this was followed by ventricular tachycardia. The rate was 422 per minute. This terminated in ventricular fibrillation and death, within a few minutes. The heart was not examined.

Dog 41–291. Three hours after operation, P_3 became higher and notched, and upper nodal rhythm developed. There was, in addition, slight depression of R–T_2 and R–T_3. Six days later upper nodal rhythm was noted. Thirteen days after operation regular sinus rhythm was present, and was noted again on the twenty-seventh day. On this later date the P–R interval was shortened (0·07 sec.).

The animal was sacrificed on the thirtieth day and organizing infarcts were found in the right auricle and almost the entire right ventricle. The epicardium over these areas was thick, opaque, and pale grey, and there were numerous dense fibrous adhesions to the pericardium. The auricle was filled with a dark-red mural thrombus which on section showed well-developed organization.

Dog 41–305. Twenty-four hours after operation, upper nodal rhythm was observed with transient left bundle branch block. On the tenth day after operation sino-auricular block was present and four days later the cardiac mechanism was normal.

The animal was sacrificed on the fifteenth day. There was recent infarction of the right auricle, which was dark reddish-purple and covered with friable, yellowish-grey granular exudate. A moderately firm dark-red thrombus filled the auricle and was tightly adherent to the endocardium. Microscopic examination showed a recent hæmorrhagic infarct with mural thrombosis. A large recent infarct of the right ventricle was also present.

The infarcts produced in the two dogs which survived were identical with the human lesion. The auricle was involved in both cases. There was massive necrosis of myocardium and endocardium and there were organizing mural thrombi (Fig. 3).

Heat and Chemical Necrosis of the Atria

Heat cauterization of right and left atria. The inferior surface of the left auricle was cauterized and P_1 became diphasic and inverted. The superior surface of the left auricle was next cauterized and P_1 became isoelectric. After cauterizing the area below the left auricle and anterior to the pulmonary veins, P_1 remained isoelectric. The entire left atrium was then cauterized and ventricular tachycardia resulted. The right atrium was cauterized with no further changes and the experiment was terminated by ventricular fibrillation.

The lateral aspect of the right auricle was cauterized without causing any electrocardiographic changes. After cauterizing the medial aspect of the right auricle, no cardiographic change was noted. Following cauterization of the entire right auricle, auricular extrasystoles occurred. The body of the right atrium was burned and slight P–Q depression resulted. At this point bleeding occurred from the necrotic wall, so that a clamp was placed across it, following which the electrocardiogram showed A-V nodal rhythm. After cauterization

of the right atrium just above the vena cava the nodal rhythm persisted. The experiment was terminated by ventricular fibrillation and hæmorrhage from the atrium.

Phenol cauterization of right atrium. Liquified phenol was applied to the entire surface of the right atrium. The heart rate was decreased from 178 to 130 per minute. The mechanism changed from normal to upper nodal rhythm. P_2 and P_3 became diphasic, and the P–R interval shortened from 0·09 to 0·06 sec. The dog died about one hour after conclusion of the experiment.

Discussion

A summary of the electrocardiographic changes following ligation of the atrial arteries and necrosis of the atria is found in Table II. No constant electrocardiographic patterns were noted, but the most frequent abnormalities were depression of P–Q, variations in the contour of P, wandering pacemaker and A-V nodal rhythm. As a control the electrocardiograms of 38 normal

TABLE II

Summary of Electrocardiographic Findings in Experimental Atrial Infarcts in Dogs

Electrocardiographic Alteration	Frequency of Occurrence	Arteries Ligated
Transient increase in amplitude and contour of P wave	11	*s**—9 animals *a**—3 animals
Slight depression of P–Q interval ..	4	*s*—1 animal All right—1 animal Heat cautery of R.A.—1 animal
Wandering pacemaker..	4	*a*—4 animals
Transient nodal rhythm	3	*s*—1 animal *a*—1 animal Heat cautery R.A.—1 animal
Transient nodal rhythm and auricular extrasystoles	3	All right and all left—1 animal All right—1 animal Chemical necrosis of R.A.—1 animal
Transient auricular tachycardia ..	1	*s*—1 animal
Auricular flutter	1	All right—1 animal
A-V block	1	All right—1 animal
A-V nodal rhythm and transient left bundle branch block sino-auricular block	1	Right coronary artery ligated near origin
P_3 notched Wandering pacemaker $R–T_2$, $R–T_3$ slightly depressed 6 days; after the operation upper nodal rhythm	1	Right coronary artery ligated near origin

* *s*, right anterior and *a*, left anterior, coronary arteries.

dogs were studied. In nine dogs there was conspicious depression of P–Q, and there were a few instances of A-V nodal rhythm and wandering pacemaker.

The electrocardiographic findings in experimental injury of the atria were somewhat comparable to the changes in clinical infarction. It is interesting to note the absence of auricular fibrillation in experimental injury, although flutter and auricular tachycardia were each present once.

During the course of these experiments a case of naturally occurring infarction of the right auricle was observed in one of the animals in the dog colony of the Institute of Pathology (Fig. 3). This colony has been in existence for twelve years and has included one thousand animals. Eight hundred and fifty autopsies have been performed, and Dr. Harry Goldblatt, who has carefully examined the hearts, says he has never seen another case of spontaneous infarction of the atrium. This dog was being used in the course of other experiments in which blood had been injected into the media of the femoral arteries and thrombophlebitis had developed. Although the right coronary artery was carefully examined no obstruction was found. The infarct resembled the human lesions because of the presence of a large mural thrombus over the area of necrosis, because of its situation, and because there were no zones of intact myocardium.

The preponderance of infarction in the right auricle of man, which has been noted in this series as well as in reported cases, may be explained by the fact that when thrombosis occurs in the right coronary artery it is usually in the first two or three centimeters, thereby occluding the atrial branches that usually supply the auricle and A-V node, although the left anterior atrial artery may also supply the latter area.

Curiously, while thrombosis of the left coronary artery and its branches occurred in 65 per cent of the cases in this series, the greatest incidence of atrial infarction was on the right side. This suggests that there may be other factors beside arterial thrombosis that will cause atrial infarction. Thus the nutrition of the atrial musculature, which may come in part through the Thebesian vessels or through the thin syncitium of the myocardium itself, may be interfered with by a large mural thrombus in the auricle. The high oxygen content of the arterial blood of the left atrium may prevent frequent infarction on this side.

We have been especially impressed with the fact that most of the cases of human atrial infarction reported in this paper have shown abnormalities of the auricular mechanism in the electrocardiograms. There have been auricular extrasystoles, auricular fibrillation, wandering pacemaker, nodal rhythm, and in five cases depression of the P–Q interval.

Summary

Infarction of the cardiac auricles (atria) was found in 17 per cent of 182 cases of myocardial infarction that were proven at autopsy. Abnormalities in the auricular complex of the electrocardiogram were present in 74 per cent of the cases of atrial infarction, but in only 9 per cent of all cases of

infarction of the ventricles. Ligation of the atrial arteries in dogs produced abnormal auricular mechanism in only 6 out of 20 experiments. In 4 additional experiments there was a transient change in the contour of the P wave. Depression of the P–Q segment of the electrocardiogram was seen in 4 instances in which the atrial arteries were ligated. Abnormality in auricular mechanism is the most reliable clue to the clinical diagnosis of infarction of the atria.

References

Abramson, D. I., Fenichel, N. M., and Shookhoff, C. (1938). *Amer. Heart J.*, **15,** 471.
Bean, W. B. (1938). *Ann. intern. Med.*, **12,** 71.
Clerc, A., and Levy, R. (1925). *Bull. Mém. Soc. méd. Hôp. Paris*, **49,** 1603.
Clowe, G. M., Kellert, E., and Gorham, L. W. (1934). *Amer. Heart J.*, **9,** 324.
Condorelli, L. (1929). *Z. ges. exper. Med.*, **68,** 493.
Davenport, A. B. (1928). *Amer. J. med. Sci.*, **176,** 62.
Feil, H., Cushing, E. H., and Hardesty, J. T. (1938). *Amer. Heart J.*, **15,** 721.
von Glahn, W. C. (1936). *Diseases of the Coronary Arteries and Cardiac Pain.* The MacMillan Company, New York, p. 143.
Gross, L. (1921). *The Blood Supply to the Heart.* Paul B. Hoeber, New York.
Hahn, L., and Langendorf, R. (1939). *Acta med. Scand.*, **100,** 279.
Krumbhaar, E. B., and Crowell, C. (1925). *Amer. J. med. Sci.*, **170,** 828.
Laignel-Lavastine, Liber, A. F., and Bidou, S. (1934). *Arch. Mal. Cœur*, **27,** 580.
Lambert, J. (1937). *Ibid.*, **30,** 3.
Langendorf, R. (1939). *Acta med. Scand.*, **100,** 136.
Lisa, J. R., and Ring, A. (1931). *J. Lab. clin. Med.*, **16,** 1083.
Meek, W. J., Keenan, M., and Theisen, H. J. (1928–29). *Amer. Heart J.*, **4,** 591.
Sanders, A. (1939). *Amer. J. med. Sci.*, **198,** 690.
Shipley, R. A., and Hallaran, W. R. (1936). *Amer. Heart J.*, **11,** 325.

PROGNOSIS OF RIGHT BUNDLE BRANCH BLOCK: A STUDY OF 104 CASES*

BY

GEORGE A. PERERA, SAMUEL A. LEVINE, AND HERMAN ERLANGER

* *From the Medical Clinic of the Peter Bent Brigham Hospital, U.S.A.*

Received December 29, 1941

Prompted by the clinical impression that patients with right bundle branch block have a better prognosis than do those with cardiographic evidence of a left bundle lesion, a study of a group of cases with the former disorder has been prepared with particular reference to the survival time following diagnosis.

The first description of the electrocardiographic manifestations of bundle branch interruption in 1910 by Eppinger and Rothberger and later by Lewis in 1916 was followed by a period of classification. The criteria of Carter (1914) were rightfully questioned by numerous observers (Oppenheimer and Rothschild, 1917; Oppenheimer and Pardee, 1920; Fahr, 1920; & Mann, 1920), until Barker, Macleod, and Alexander (1930) presented substantial proof (supported later with Wilson, 1932) that the common type of bundle branch block was left, and not right as was formerly thought; and that the unusual type of block was in reality due to a defect in the right bundle branch. In 1934 Bayley divided the curves of right bundle branch block into four clearly differentiated groups.

In recent years it has been suggested that a deviation of the electrical axis plays a role in the formation of the characteristic curves of bundle branch block (Prinzmetal, Oppenheimer, and Dack, 1937), and that the type of block may depend on which ventricle is predominantly enlarged (Master, Kalter, Dack, and Jaffe, 1940). Even the careful review and histopathological analysis of Yater (1938) leaves doubt as to exact clinical correlations. One is forced to admit that the diagnosis of right or left bundle branch block is purely electrocardiographic, cannot be foretold by examination of the patient, and cannot always be determined by microscopic section. Furthermore, the exact mechanism whereby these curves are produced is not completely understood.

Our Material and Results

One hundred and four cases of right bundle branch block were selected from private records and from the house and out-patient departments of the Peter Bent Brigham Hospital (60 and 44 cases respectively) on the basis of their

* An abstract of this paper was presented before the New England Heart Association at Boston, January, 1941.

fulfilment of electrocardiographic criteria of diagnosis. Cases of bundle branch block with short P–R interval (Wolfe-Parkinson-White syndrome), which have a good prognosis, were excluded. Serological data were available in about one half the group, and on the charts of three-quarters specific answers were given to questions concerning a previous history of rheumatic fever or its manifestations. Follow-up information was secured in some instances by letters from either the patient or his family physician, and in the remainder by our own examination.

All cases with a QRS interval of 0·12 sec. or over and a conspicuous S wave in lead I (Wilson, Johnston, Hill, and Macleod, 1934; & Wilson, Johnston, and Barker, 1934) were selected; all others were discarded. Although the T waves were upright in lead I and inverted in lead III in all but 4 instances, and increased amplitude of the QRS deflections was present in all but 6 cases, these were not used as diagnostic criteria.

Sex and Age.—The average age of the 104 patients was 56 years. Of these, 75 were male and 29 were female. The men averaged 55 and the women 58 years of age. The age and sex distribution are illustrated in Fig. 1. It is

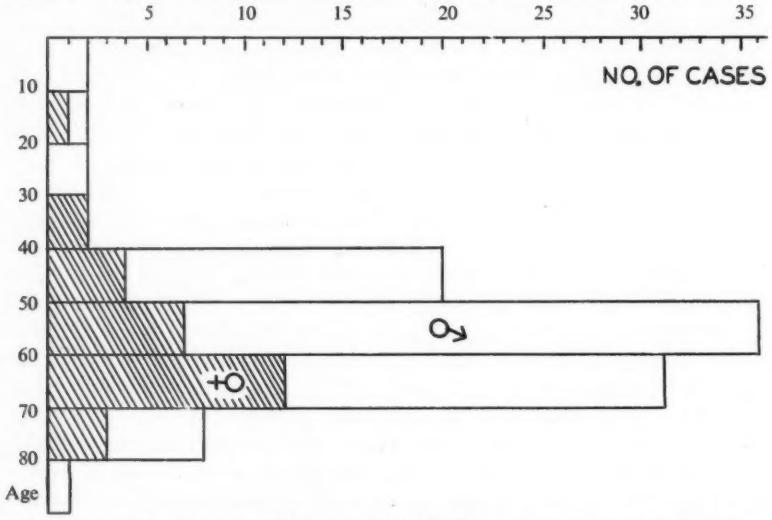


FIG. 1.—Age distribution of 104 cases of right bundle branch block when the diagnosis was first made, males and females.

of some interest that the youngest was 5 years old, and that there were four under 20 years of age.

Antecedent Cardiac Symptoms.—Before the diagnosis of right bundle branch block by electrocardiogram, 9 patients denied any symptoms or signs referable to the heart; in fact the condition in these cases was entirely symptomless. The average duration of antecedent symptoms in the entire group was two years and nine months. Of those with cardiac manifestations (95 of the 104 cases) major complaints were as follows: 53 had dyspnœa; 39, angina

pectoris; 15, præcordial (but not anginal) pain; 12, palpitation; 10, syncopal attacks; and 8, a history of dizziness. As the presenting complaint, angina pectoris was the chief difficulty in 30, dyspnœa in 27 cases.

Congestive Failure.—On examination at the time of diagnosis, 77 patients showed no signs of right- or left-sided cardiac insufficiency, 14 showed slight evidence of failure, 12 moderate evidence, and only 1 severe congestive failure. The comparative benignity of right bundle branch block becomes evident from these figures.

Cardiac Enlargement.—32 had a slight increase and 18 showed enlargement well beyond the left mid-clavicular line. In 9 the cardiac borders could not be determined. There was no enlargement of the cardiac area by percussion in 40. In 11 of the 24 patients, who had teleo-radiograms taken, an increase in the size of the heart was apparent.

Blood Pressure.—The following figures exclude the determinations obtained in the 3 patients who were *in extremis* when first seen. The average blood pressure of 101 patients was 155 mm. systolic, and 88 mm. diastolic. 59 patients had systolic pressures over 140; these ranged up to 240 mm., the average being 178 mm. ; the diastolic readings were nearly all greater than 90 mm., and ranged from "0" to 165 mm., the average being 96 mm. Of these hypertensive cases, 38 (64 per cent) were males, the remaining 21 (36 per cent) being females. 42 patients had systolic readings of from 100 to 140 mm., the corresponding diastolic pressures being from 60 to 90 mm. Of these cases, 36 (86 per cent) were males, while the remaining 6 (14 per cent) were females. It is apparent that the blood pressure in these cases may vary from low to extremely high levels, though on the average it is slightly elevated.

Auscultation of the Heart.—Reduplicated heart sounds, gallop rhythm, and pulsus alternans are often regarded as bearing some relationship to bundle branch block. It is of interest that gallop rhythm was noted in but 10 instances, reduplicated sounds heard in 6, and pulsus alternans present in but 5. Except in one instance, all patients having a protodiastolic gallop or alternating pulse showed signs of advanced cardiac disease and death occurred within two years. A specific notation as to the presence or absence of these signs was made in 80 per cent of the series. The most common auscultatory abnormalities were an apical systolic murmur (24 patients) and distant heart sounds (20 patients).

QRS Interval.—68 cases had a QRS interval of 0·12 sec., 18 of 0·13 sec., 14 of 0·14 sec., 3 of 0·15 sec., and 1 case an interval of 0·16 sec.

All of the patients with right bundle branch block in this series fitted into one of the four groups described by Bayley in 1934 (Fig. 2). This investigator classified 70 cases with right-sided bundle branch lesions, and found that 20 per cent were in group I, 33 per cent in group II, 40 per cent in group III, and 7 per cent in group IV. Our findings were very similar with 23 per cent in the first group, 28 per cent in the second, 45 per cent in the third, and 4 per cent in the fourth category.

Associated Electrocardiographic Abnormalities.—As regards disorders of rhythm, premature ventricular beats were demonstrated in 8 cases, premature

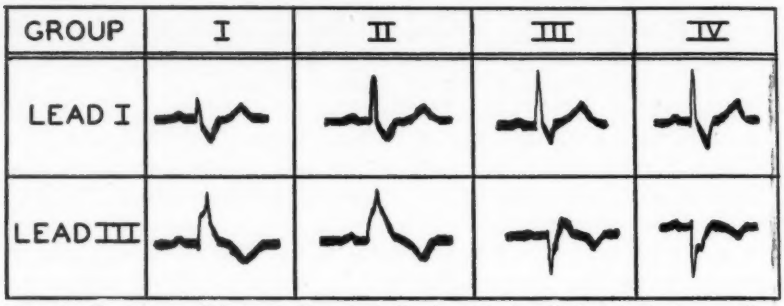


FIG. 2.—Four types of electrocardiograms indicating right bundle branch block, according to Bayley classification.

auricular contractions in 4, auricular fibrillation in 4, and sinus pauses in 2. Conduction disorders were found in 9 patients: delayed auriculo-ventricular conduction in 4, incomplete A-V block with dropped beats in 3, and intermittent complete block in 2. Low voltage was recorded in 6 cases.

Other changes appeared only in subsequent electrocardiograms. Two patients developed auricular fibrillation some time after the original diagnosis of bundle branch block, one partial block became complete, one with regular rhythm showed a complete block, and in one instance there was lengthening of the P–R interval.

Ætiology.—Hypertensive cardiovascular disease was the underlying cause in 48 patients. Degenerative heart disease was the cause in 40: it was diagnosed by the presence of cardiac signs or symptoms in the absence of hypertension, valvular disease, or other obvious cause. Rheumatic heart disease with damaged valves were the cause in 9, congenital defect in 2, and syphilitic aortitis with aortic insufficiency in 1 patient. In 4 patients no definite diagnosis was established: one was a young man without apparent illness, the second had gout, the third pneumonia, and the fourth had myxœdema.

A clinical diagnosis of myocardial infarction was made, either before or after the appearance of the bundle branch lesion in 10 patients. Other associated diagnoses included diabetes in 6, asthma in 3, and carotid sinus sensitivity in 2 patients. One of the hypertensive group had a toxic nodular goitre during the course of his illness.

Follow-Up.—Of the 104 in this series, 41 were capable—during the period of observation—of carrying out their normal activities without limitation because of cardiac symptoms. 13 patients were seen at the time of diagnosis only, and no subsequent data could be obtained. Of the 91 patients followed, 62 were living an average of four years and one month later, while 29 died within an average of three years. 29 patients were known to have died, 4 of cardiac failure, 3 of coronary occlusion, and 2 of cerebral hæmorrhage. 9 died of unrelated causes, and in 11 cases information as to cause of death was inadequate. A more detailed analysis is shown in Table I.*

* In the period during which this manuscript was being prepared for publication, Willius, Dry, and Reeser (1941, *Arcn. intern. Med.*, **67**, 1008, *ibid.*, **67**, 1027, *ibid.*, **67**, 1034) have in general confirmed these results.

TABLE I

THE PROGNOSIS OF PATIENTS WITH RIGHT BUNDLE BRANCH BLOCK

	PATIENTS ALIVE AT END OF					
	1 year.	2 years.	3 years.	4 years.	5 years.	10 years.
62 patients alive when last followed ..	52 (84%)	44 (71%)	33 (53%)	25 (40%)	19 (31%)	5 (8%)
29 patients known to have died ..	18 (62%)	15 (52%)	11 (38%)	9 (31%)	7 (24%)	1 (3%)
23 patients known to have died * ..	18 (77%)	15 (65%)	11 (48%)	9 (39%)	7 (30%)	1 (4%)
91 total followed patients	70 (77%)	59 (65%)	44 (48%)	34 (37%)	26 (29%)	6 (7%)

* This group omits 6 cases that died within a few days of first examination.

Six patients (21 per cent of the group followed until death) died within a few days of the original diagnosis. One patient, with angina pectoris, died of metastatic neoplasm sixteen years and seven months after the discovery of right bundle branch block, and another was known to be alive after seventeen years. Patients under 50 years of age lived an average of five years, while those aged 50 or over averaged just under three years.

PROGNOSIS

When one speaks of the prognosis of patients with bundle branch block, it must be realized that the lesion may have ante-dated its electrocardiographic demonstration by an indefinite period. Furthermore, it might be thought that the average survival time amongst the group still living, if followed to death, would be decidedly greater than that of the known fatal cases. This does not prove to be so in studies of prognosis when the number of cases analysed is sufficient; the main reason, of course, being that amongst the living cases there are some, who have only recently been observed for the first time and may yet die within a short period, to compensate for those already known to be alive for a great many years. In fact, Table I shows very little difference between the survival time of the fatal and non-fatal cases.

Attempts at comparison with other follow-up studies of bundle branch block show that diagnostic criteria were not always identical, presentation of analyses were not always similar, and in some series, electrocardiograms were taken routinely, while in others, only in suspected heart disease. In addition, as this disorder of conduction has only been known for a short period of time, early investigations included a smaller number of cases, shorter survival observations, and fewer cases followed until the fatal termination.

Early studies (Carter, 1914; Willius, 1919; Herrick and Smith, 1922;

Hart, 1925; Cowan and Bramwell, 1925; Talley and Reed, 1926; Bach, 1930; Campbell and Turkington, 1931; Graybiel and Sprague, 1933; & King, 1934) included cases with QRS intervals of 0·11 sec., often failed to separate right and left types or added indeterminate forms of bundle branch block. In almost all such series, consisting chiefly of cases of left-sided disorders, the majority of patients were dead six to fourteen months after diagnosis. Von Deesten and Dolganos (1934) were among the first to comment on the favourable prognosis of right bundle branch block (5 cases), and the 64 cases presented by Wood, Jeffers, and Wolferth (1935) did unusually well.

On the other hand, Sampson and Nagle (1936) reported that only 27 per cent of 109 cases of left bundle branch conduction defect that terminated fatally were dead in the first year and 42 per cent by the second year, although they included those with a QRS interval of 0·11 sec. Freund and Sokolov (1939), using adequate diagnostic criteria, claimed that the average length of life after discovery of the lesion was about the same (one year) in 58 fatal cases of left block as in 32 cases of right bundle branch block. Bishop and Carden (1939) found the average survival time of 29 cases that died was three years, but intermediate and a few right-sided lesions were included. Kaplan and Katz (1939), again without subdivision as to types, stated that 80 per cent of 75 known fatal cases died during the first year.

It is evident from the preceding that a great deal of variation exists in prognostic methods and results, but that only the left bundle branch block series of Sampson and Nagle (1936) and Bishop and Carden (1939) show longer survival periods than do the cases of right bundle branch block presented in this study. It is also true that other reports of right bundle branch block are not consistent, Wood and his associates (1935) differing widely from Freund and Sokolov (1939) in their respective studies. Yet it is unfair to place the majority of these follow-up series side by side because of difference in diagnostic criteria, admixtures of indeterminate types of block, and dissimilar methods of statistical analysis.

For this reason a group of 60 patients with left bundle branch block (with QRS interval of 0·12 sec. or over, and an upward deflection lead I without Q or S waves), followed until death, were selected for comparison. This series was prepared so that the ratio of private and hospital cases and the dates of the original diagnosis were similar to the 29 fatal cases reported above. The average age of this group was 59 years. The results (Fig. 3) show decidedly more favourable survival figures in patients with a right bundle branch conduction defect. The average time of death after diagnosis of the left bundle group was one year and two months, with 60 per cent of the cases dead by the end of the first year. The longest survival time in this fatal group was five years and eight months. One patient not included in this study, because he is still alive, is in fairly good health fifteen years after left bundle branch block was first discovered.

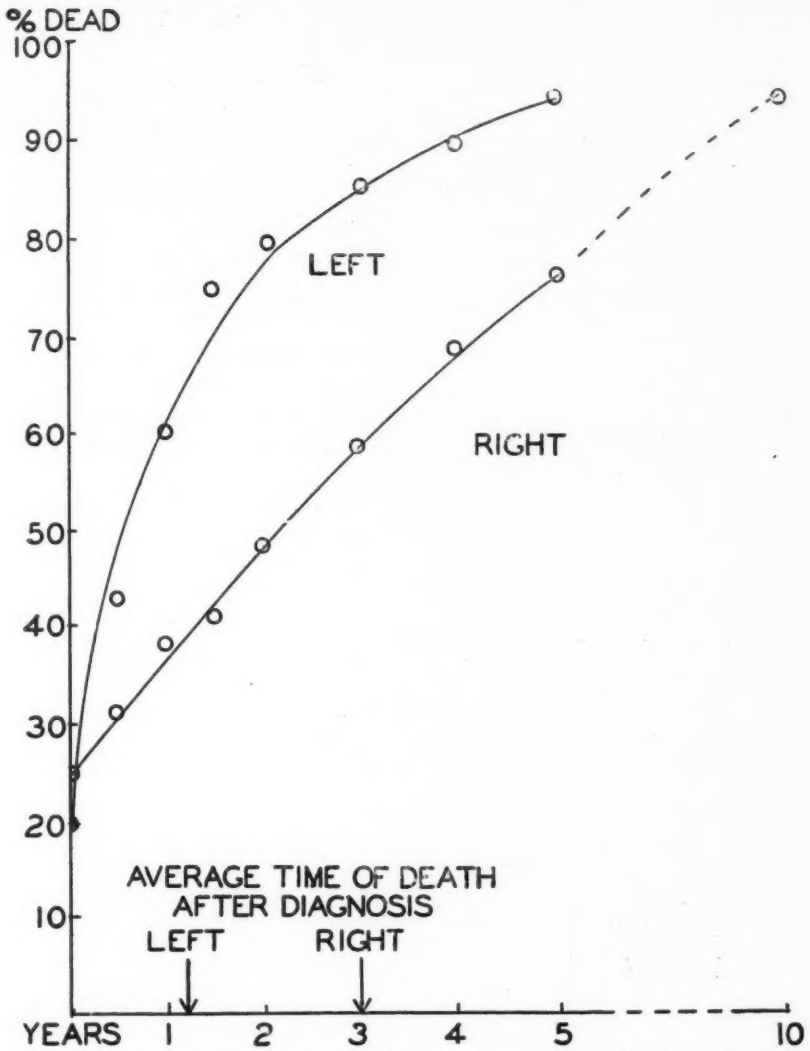


Fig. 3.—Comparison between the survival period of cases of left and right bundle branch block.

Conclusions

An analysis of 104 cases of right bundle branch block has been prepared.

It was observed that reduplicated heart sounds, gallop rhythm, and pulsus alternans were uncommon in this series. The two latter disorders were noted in a small group that had advanced heart disease.

Forty per cent of the patients had no appreciable subjective discomfort from the heart. In fact, some have no subjective or objective evidence of heart disease apart from the right bundle branch block.

The average survival time after the diagnosis of right bundle branch block of 29 fatal cases was three years. If the 6 cases that came into the hospital more or less moribund and died within a few days are excluded, the average survival period of the fatal group was four years and five months. Of the 62 patients alive when last seen or heard from, the average survival period was four years and one month. The longest survival period amongst the living cases was seventeen years, and amongst the fatal cases, sixteen years and seven months.

The clinical impression that patients with right bundle branch block have a distinctly more favourable prognosis than do those with a left bundle branch lesion has been sustained.

References

Bach, F. (1930). *Quart. J. Med.*, **23,** 261.
Barker, P. S., Macleod, A. G., and Alexander, J. (1930). *Amer. Heart J.*, **5,** 720.
Bayley, R. H. (1935). *Amer. J. med. Sci.* **188,** 236.
Bishop, L. G., Jr., and Carden, G. A., Jr. (1939). *Amer. Heart J.*, **17,** 275.
Campbell, S. B. B., and Turkington, S. I. (1930). *Quart. J. Med.*, **24,** 481.
Carter, E. P. (1914). *Arch. intern. Med.*, **13,** 803.
Cowan, J., and Bramwell, J. C. (1925). *Quart. J. Med.*, **19,** 95.
Eppinger, H., and Rothberger, C. J. (1910). *Z. klin. Med.*, **70,** 1.
Fahr, G. (1920). *Arch. intern. Med.*, **25,** 146.
Freund, H. A., and Sokolov, R. (1939). *Ibid.*, **63,** 318.
Graybiel, A., and Sprague, H. B. (1933). *Amer. J. med. Sci.*, **185,** 395.
Hart, T. S. (1925). *Arch. intern. Med.*, **35,** 115.
Herrick, J. B., and Smith, F. M. (1922). *Amer. J. med. Sci.*, **164,** 469.
Kaplan, L. G., and Katz, L. N. (1939). *Amer. Heart J.*, **18,** 145.
King, J. T. (1934). *Amer. J. med. Sci.*, **187,** 149.
Lewis, T. (1916). *Phil. Trans. Roy. Soc., B,***207,** 221.
Mann, H. (1920). *Arch. intern. Med.*, **25,** 283.
Master, A. M., Kalter, H., Dack, S., and Jaffe, H. L. (1940). *Amer. Heart J.*, **20,** 186.
Oppenheimer, B. S., and Rothschild, M. A. (1917). *J. Amer. med. Ass.*, **69,** 429.
——, and Pardee, H. E. B. (1920). *Proc. Soc. exper. Biol. and Med.*, **17,** 177.
Prinzmetal, M., Oppenheimer, B. S., and Dack, S. (1937). *J. Amer. med. Ass.*, **108,** 620.
Sampson, J. J., and Nagle, O. E. (1936). *Amer. J. med. Sci.*, **191,** 88.
Talley, J. E., and Reed, O. K. (1926). *Amer. Heart J.*, **1,** 262.
von Deesten, H. F., and Dolganos, M. (1934). *Amer. J. med. Sci.*, **188,** 231.
Willius, F. A. (1919). *Arch. intern. Med.*, **23,** 431.
Wilson, F. N., Macleod, A. G., and Barker, P. S. (1932). *Amer. Heart J.*, **7,** 305.
——, Johnston, F. D., Hill, I. G. W., Macleod, A. G., and Barker, P. S. (1934). *Ibid.*, **9,** 459.
——, ——, and Barker, P. S. (1934). *Ibid.*, **9,** 472.
Wood, F. C., Jeffers, W. A., and Wolferth, C. C. (1935). *Ibid.*, **10,** 1056.
Yater, W. M. (1938). *Arch. intern. Med.*, **62,** 1.

DR. SAMUEL JOHNSON:

HIS MEDICAL HISTORY AS RECORDED BY JAMES BOSWELL

BY

TERENCE EAST

Received February 14, 1942

The medical man who reads Boswell's *The Life of Samuel Johnson, LL.D.* with a professional eye will find therein the history of a case of high blood pressure, the course of which can be traced through many familiar phases.

Johnson died at the age of seventy-five, on December 13, 1784. " His person was large and robust, I may say approaching the gigantic." To us nowadays, by a stroke of irony which he might have appreciated, for he was a water-drinker much of his life, " his countenance of the cast of an ancient statue " has been made familiar by an advertisement for beer ! His face was scarred with scrofula, for which, at the age of thirty months, he had been taken to Queen Anne to be touched for the King's Evil; he remembered her dimly in later years as a " lady in diamonds and a long black hood." At the age of sixty-four, in spite of his unwieldy frame—for " when he walked it was like the struggling gait of one in fetters ; when he rode, he had no command or direction of his horse, but was carried as if in a balloon "—he undertook the " Tour of the Hebrides " : no small journey in those days for a man of his age, even for one in good health. But as by many, whose tissues are good, the high blood pressure was well tolerated, at first. His parents had lived long, his father dying at seventy-eight and his mother at ninety.

Gout, high blood pressure, and arteriosclerosis are closely associated, and it is with an attack of gout at the age of sixty-five that we see the first indications of the illness that troubled Johnson's last years. It is not surprising to find the ". . . pangs arthritic that infest the toe of libertine excess " as Cowper* called them, so prevalent in the gluttonous eighteenth century, in one whose appetite can only be described as voracious.

Two years later, at sixty-seven, he writes to Boswell of another attack: " I was seized with gout, and am not quite well. The pain has not been violent, but the weakness and tenderness were very troublesome, and what is said to be very uncommon, it has not alleviated my other disorders." The idea of gout relieving other disorders was widely held; and one can recall several true examples of intercurrent infection curing another disease. The other disorders to which Johnson refers are, no doubt, " a difficult and laborious respiration,"

* *The Task.*

which Boswell had already noted with alarm and foreboding; for which he " lost six and thirty ounces of blood in a few days," apparently with some benefit.

Next year Johnson writes: " The hand of time or disease is heavy upon me. I pass restless and uneasy nights, harassed with convulsions of my breast, and flatulencies of my stomach." Perhaps the gallstone that was found at autopsy was responsible for some of this gastric derangement. At the age of seventy-one he was " hindered by a vexatious and incessant cough," the treatment for which he describes thus to his friend and physician, Dr. Lawrence: " bled once, fasted four or five times, taken physick five times " (purges, no doubt) " and opiates, I think, six "—sound treatment for such a man, and perhaps fasting, in these days, might be recommended for plethoric patients more often.

On his birthday, however, Johnson felt that he was beginning the seventy-second year of his life with more strength of body and greater vigour of mind than, he thought, was common at that age. This is about the last reference to good health that we find. In the early part of next year he complains again of his bronchitis: " a troublesome time with my breath; and a violent cold," for which he was " let blood three times." A " catarrhous cough " still harassed him in June. Twelve months later, when he was seventy-four, Boswell comments: " My anxious apprehensions proved to be too well founded," for Johnson had a stroke. His vivid description of this attack is given in a letter to his old friend, Mrs. Thrale, two days later.

" I went to bed, and in a short time waked and sat up, as has long been my custom, when I felt a confusion and indistinctness in my head, which lasted, I suppose, about half a minute. I was alarmed, and prayed God that however He might affect my body, He would spare my understanding. This prayer, that I might try the integrity of my faculties, I made in Latin verse. The lines were not very good, but I knew them not to be very good: I made them easily, and concluded myself to be unimpaired in my faculties. Soon after I perceived that I had suffered a paralytic stroke, and that my speech was taken from me." For such a one as Johnson, this was perhaps the most severe deprivation that he could have suffered. But he was so little upset, " that I wondered at my own apathy."

He at once sent for his neighbour, Dr. Brocklesby, a Quaker, practising near the Strand, and for his " dear Dr. Heberden," of whom the poet Cowper * wrote,

> " Virtuous and faithful Heberden, whose skill
> Attempts no task it cannot well fulfill."

By July, he was able to write: " The physicians consider me cured, and I had leave four days ago to wash the cantharides from my head." He was sufficiently recovered to pay several visits in the country.

It seems likely that this attack was a transient paresis due to cerebral arteriospasm, of the type commonly met with in hypertensive encephalopathy.

In September he was again attacked by gout, which, he writes, " has within

* *Retirement, Satirical and Didactic Poems.*

these four days come upon me with a violence which I had never experienced before." At the end of the year Boswell records: " He was seized with a spasmodic asthma of great violence, being obliged to sit up all night in his chair, a recumbent posture being so hurtful to his respiration that he could not endure lying in bed." Such asthma was surely what we call cardiac: bronchial asthma would hardly appear for the first time at his age. This development of nocturnal dyspnœa was ominous; and the gravity of the outlook was increased by " that oppressive and fatal disease, a dropsy." In those days, one may remark, dropsy was regarded as a disease *sui generis*; it was not recognized as a result of failure of the heart.

Johnson had entered upon the last year of his life. The first attack of dyspnœa seems to have come on very suddenly on exertion. Early in December he was going to the opening meeting of a new club that he had founded, the " Essex Head," in Essex Street when, he writes: " I was seized with a spasmodic asthma, so violent that with difficulty I got to my own house, in which I have been confined eight or nine weeks, and from which I know not when I shall be able to go even to Church. A dropsy gains upon me; my legs and thighs are very much swollen with water." Anxious over these symptons, and " extremely afraid of dying," he wrote to Boswell to ask him to obtain the opinions of the physicians in Edinburgh on his case. Before the advice came he seems to have already gained some " unexpected relief by the discharge of twenty pints of water." Perhaps this was due to the vinegar of squills which he had begun to take. Boswell wrote an excellent summary of his case, which he communicated to the great Edinburgh physicians, Cullen, Hope, and Monro. He noted the sense of præcordial constriction on exposure to cold (this winter was a very severe one), and mentioned the tincture of laudanum Johnson took for his nocturnal dyspnœa. But what the doctors advised is unfortunately not recorded.

Johnson was interested in his symptoms, and had read Sir John Floyer's book on asthma. One may recall that Floyer was for many years, until his death in 1734, physician at Lichfield, the birthplace of Johnson, and possibly may have attended him as a child and advised his being touched by Queen Anne. Writing to his friend Langton in March, 1784, he remarks: " My asthma is, I am afraid, constitutional and incurable; but it is only occasional, and unless it be excited by labour, or by cold, gives me no molestation, nor does it lay any close siege to life, for Sir John Floyer, whom the physical race consider as author of one of the best books upon it, panted on to ninety as was supposed." From this one gathers that Johnson thought his case might have a similar prognosis. But he was quoting without the book. During the spring and summer he was free from dropsy, and was able to take a jaunt to Oxford in June, and thence on to Lichfield for five days. Here, on July 19, he apparently borrowed Floyer's text book from the Cathedral library* for further information about his asthma, which seems to have returned. The conclusion was unsatisfactory. Writing to Dr. Brocklesby the next day he reported that " The asthma has no abatement. Opiates stop the fit so that I can sit and sometimes lie easy . . ., I am looking into Floyer, who lived with his asthma till almost

* *Floyer*, by J. A. Gunn, in *British Masters of Medicine*.

his ninetieth year. His book by want of order is obscure; and his asthma, I think, not of the same kind with mine." In which deduction he was correct, for Floyer had bronchial asthma all his life, and Johnson's was certainly cardiac.

This holiday and its travelling in coaches was too much for his heart. The increasing dyspnœa was followed by a return of dropsy in the middle of August. It is interesting to note that in one letter he says: " My respiration gets more and more ease and liberty. I went to Church yesterday after a very liberal dinner, without any inconvenience," while a few lines further on he remarks: " the water rises, though it does not rise very fast." Evidently the relief of the dyspnœa occurred when the failure of the right ventricle began to ease the state of his pulmonary circulation, embarrassed by a failing left ventricle. " The squills I have not neglected; for I have taken more than a hundred drops a day, and one day took two hundred and fifty, which, according to the popular equivalent of a drop to a grain, is more than half an ounce." Thanking his physician for his attention, he pays him the neat compliment: " If the virtue of the medicines could be enforced by the benevolence of the prescriber, how soon should I be well." But he could be critical enough of what was prescribed if he thought it of no value. Perhaps something other than squills had been suggested. " I never," he writes to Brocklesby, " thought well of Dr. James's compounded medicines." One may recall that Dr. James patented a famous febrifuge powder of phosphate of lime and oxide of antimony. Johnson esteemed him, for he wrote a dedication to Dr. Meade in his book, and said of him, " no man brings more mind to his profession "—" This prescription exhibits a composition of about three hundred and thirty grains, in which there are four grains of emetic tartar, and six drops of thebaick tincture." (This was tincture of opium, for opium was prepared at Thebes). " He that writes thus surely writes for show. The basis of his medicine is the gum ammoniacum " (an expectorant from dorema ammoniacum), " which dear Dr. Lawrence used to give, but of which I never saw any effect. We will, if you please, leave this medicine alone. The squills have every suffrage, and in the squills we will rest for the present." His critical mind detected the shams of the mediæval polypharmacy still much in vogue, and recognized a potent drug when he tried it. As a result, by October the failure seems to have abated, for he could write that he was relieved from the dropsy. After visiting Birmingham, and Oxford again, he was back in London by the middle of November. The journeys seem to have brought on heart failure again, for he writes: " I am as I was, and having seen Dr. Brocklesby, am to ply the squills." Up to this date he had kept a Latin diary of his case, " *Aegri Ephemeris*," but it has not survived.

The end was near. The dropsy rapidly accumulated once more. In order to relieve it incisions were made in his legs by Cruikshank, the surgeon; " a sweet blooded man," as Johnson called him, but a timid operator, who did not cut deep enough to satisfy his patient, who himself " with his usual resolute defiance of pain cut deep, when he thought that his surgeon had done it too tenderly." A few days before his death he appropriately quoted to Brocklesby the lines from *Macbeth*:

" Cans't thou not minister to a mind diseased;
Pluck from the memory a rooted sorrow;
Raze out the written troubles of the brain;
And, with some sweet oblivious antidote,
Cleanse the stuff'd bosom of that perilous stuff
Which weighs upon the heart ? "

His physician at once capped the quotation by answering

" . . . therein the patient
must minister to himself."

a neat rejoinder, which pleased Johnson much, if it gave but little physical help. On hearing that he could not recover, he refused all further drugs; he wished to have his mind unclouded at the end. So he died quietly, somewhat troubled with breathlessness and pain in the legs, his mind clear, on December 13. He complained less of his dyspnœa in this, his third and last, attack of failure than at first, presumably because once again failure of the right ventricle came on fast.

The report of the post-mortem examination can be seen in the library of the Royal College of Physicians. It was done two days after death by Dr. J. Wilson; Mr. Cruikshank and Dr. Heberden being present. I quote the account. " Dr. Johnson being very importunate to have the water entirely gone, the morning of the day on which he died, repeated the operation himself, and cutting very deep. For several years he had been troubled with asthma, for which he commonly used to take opium. The lungs did not collapse as they usually do when air is admitted, but remained distended as if they had lost the power of contraction. The air cells on the surface of the lung were also much enlarged, the right lobe adhered very strongly to the diaphragm. No water was found in the cavity of the thorax. The heart was exceedingly large and strong; the valves of the aorta were beginning to ossify. No more fluid than common was contained in the pericardium. In the peritoneum there seemed to be incipient peritoneal inflammation and ascites. The liver and spleen were large and firm. The spleen had almost the feel of cartilage. A gallstone the size of a pigeon's egg was taken out of the gall bladder." He had not been much troubled by this stone apparently; very likely it was a cholesterol solitaire, which often causes little or no symptoms. The account describes also " hydatids " in the right kidney, and how the left was almost entirely destroyed, with two very large hydatids formed in its place. " Mr. White pricked his finger. The next morning he had red lines running up his arm, and a slight attack of fever."

The autopsy revealed much that we would expect. The severe degree of hypertrophic emphysema was likely to be found in a man who had suffered much from bronchitis, and it contributed a great deal to the acuteness of his dyspnœa. The great hypertrophy of the heart was no doubt the result of high blood pressure, for the early calcification of the aortic valves can hardly have amounted to actual stenosis. The liver and spleen showed the effects of chronic venous engorgement. The kidneys were of the small

gouty type, with retention cysts, but there is no hint that they were deficient enough to cause uræmia.

Summary

The medical history of Samuel Johnson is that of a man, with good family history, who had gout and developed high blood pressure, probably rather late, and then had a transient stroke. Failure of the left ventricle came on, and emphysema aggravated his symptoms. He was, for a time, successfully treated by rest, squills, and opium. But on his recovery, he undertook too much travelling and brought on failure again. He finally died in his third bout of heart failure, about a year after the onset of the first attack.

It seems natural and almost inevitable that Dr. Johnson, so loved by Englishmen for his ordinary qualities, should have died from such a common and straightforward combination of diseases.

The fact that Boswell's description enables one to deduce so much, and that it might indeed serve as a model lesson for a medical student, goes some way to explain the immense popularity of his book.

INVERSION OF T WAVES AFTER LONG PAROXYSMS OF TACHYCARDIA

BY

MAURICE CAMPBELL

From the Cardiographic Department, Guy's Hospital

Received February 4, 1942

For several years I have been struck by odd cases where changes in the T waves and even sharp inversion of these waves after long paroxysms of tachycardia—in appearance suggestive of coronary occlusion or at least of coronary disease—seemed from the subsequent course of the patient to be no more than the effect of the long paroxysm. Sometimes when these changes were observed it was difficult to exclude active though undiagnosed myocardial disease, such as might follow a silent cardiac infarct; and sometimes the patient had taken digitalis or quinidine in an effort to arrest the attack in such amounts that these might possibly have caused the T wave inversion, though considering the dosage and the short time for which the drug was taken this seemed most unlikely.

In a recent paper (Campbell, 1939) two illustrations of such changes were given with the legend " changes produced by a paroxysm, but simulating the changes after cardiac infarction."

In the first there was a fairly deep inversion of T of the " coronary " type in lead I with a correspondingly high T in lead III, two days after a paroxysm. After another two days these had both diminished, and three weeks later had returned to normal, the T wave being small but upright in lead I and flat in lead III (Fig. 21, A, B, and C, *loc. cit.*). This was from a patient who started having paroxysms after diphtheria, and appeared in good health in the intervals.

In the second there was inversion of the whole S–T interval (perhaps of a less obvious coronary type) in leads II and III, one day after a long paroxysm; eleven days later these effects had greatly diminished and eighteen days later they had completely disappeared (Fig. 22, A–D, *loc. cit.*). This was from a man, aged 40, whose attacks had been present for at least fifteen years, leaving him without any disability between times in the earlier period, but with some gradually increasing dyspnœa in the later period.

In neither case was it thought likely that the change was due to a cardiac infarct or to coronary disease—partly on clinical grounds, and partly because of the speed with which the change was reversed after the attack. But both these patients died, one suddenly and one with congestive failure, so that it was difficult to exclude with certainty coronary disease or organic myocardial

changes as the cause, though in the case in which there was an autopsy, no coronary disease was found.

The question is of real importance, because after a long paroxysm of tachycardia, a patient is likely to seek advice about the condition of his heart and this is increasingly likely to lead to an electrocardiogram being taken. When there are changes in the T waves or in the S–T interval suggestive of myocardial disease, it is most important that these signs should not be taken as definitive evidence of such disease, if in fact they are no more than the after-effects of the long paroxysm.

Special efforts were therefore made to obtain serial cardiograms shortly after long paroxysms from patients in whom the length of history of the paroxysms and the absence of any physical signs or symptoms suggesting heart disease made it almost certain that the heart was normal, except for the paroxysms.

First Case. The first patient, where we were fortunate enough to get the chance of taking such records, had started having typical paroxysms when she was 17, at first only lasting for an hour or two, every three months or so. When she came under my observation, aged 34, she was a healthy looking woman, with four children, and young for her years. Her attacks had become more frequent and lasted longer, up to 8 or even 16 hours. Since then she has been under my regular observation for another twelve years, and still, after this length of time shows no evidence of heart disease. During all this time she has averaged about ten attacks a year, lasting generally from 8 to 16 hours. Further details need not be given as a full account of her attacks was given in Table IX in the article already referred to (Campbell, 1939), and Fig. 12, 13, and 18 were taken from her. At no time has any abnormal physical sign been found, nor any evidence of heart disease except the paroxysms, though when they lasted for more than six or seven hours, she generally got anginal pain of increasing severity as they continued. Considering the rate of her attacks (about 220) she was not excessively incapacitated and had travelled some miles to see me in attacks, because in the earlier stage they could be stopped by pressure on the neck. In more recent years they have caused greater incapacity but one could hardly expect a woman at 45 to stand them as easily as at 30, and I do not think this need be taken as evidence of any heart disease. Incidentally she is a patient in whom treatment has been quite ineffective in controlling the incidence of her paroxysms—treatment with bromides, carminatives, digitalis, and quinidine having been tried. She has written to me within the last few weeks: even a period in the East End of London during the worst of the air raids had not upset her unduly, and on the whole her attacks had been less during the war than during the preceding years.

In October, 1938, we were able to get her admitted to hospital in an attack that had lasted three days. Pressure in the neck, 10 grains of quinidine sulphate, and 20 mg. of mecholyl, given intramuscularly, did not arrest the attack, which stopped after about six hours in bed.

Serial electrocardiograms after this paroxysm provided a good illustration,

with transient T_3 inversion, not unlike that seen after a cardiac infarct. The day after the paroxysm ended there was sharp T_3 inversion of the cove-shaped type (Fig. 1B) with a flattened T_2. The next day this was much less, but T_3

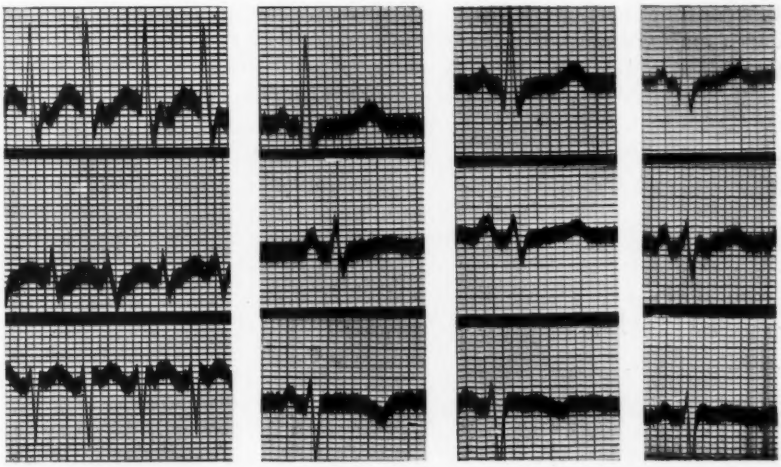


FIG. 1.—" Coronary " T wave changes in lead III, one day after a long paroxysm, in a woman with a normal heart and a history of paroxysms for many years.
(A) Paroxysm of tachycardia (6/10/38).
(B) Inversion of T in lead III, one day later (7/10/38).
(C) Diminishing inversion of T in lead III, three days later (10/10/38).
(D) Disappearance of all inversion of T in lead III, six days later (13/10/38).

was still just inverted (Fig. 1C), and three days later the inversion had completely disappeared (Fig. 1D).

It seems improbable that after some hundreds of attacks spread over 30 years one should have recorded an exceptional one that was really due to some serious coronary episode; on the contrary, as the attack to her and to other observers seemed the same as all the others it is almost certain that such changes in the T waves occurred frequently whenever an attack was of sufficient length.

Second Case. During the same month, we obtained inconclusive records from another patient (Fig. 3) in whom we suspected similar changes (Fig. 2), but within two months after a longer attack we got most satisfactory evidence in her case also (Fig. 4). This second patient, who was first seen in 1932 when she was 44, had suffered from paroxysmal tachycardia since 17 years of age. She had diphtheria at 13, but no rheumatic fever, chorea, or rheumatism. The paroxysms were characteristic, and have since been confirmed graphically (Fig. 3A). From the age of 17 to 44, she was never six months without an attack and often had two a week. In 1932 she had fourteen attacks mostly lasting between 12 to 24 hours. In general they had lasted from 12–48 hours, though

some were shorter, and one, when she was 21, was said to have lasted for two weeks, and another, when she was 35, for three weeks. Bending or stretching were liable to start attacks and she was afraid of hurrying or sudden exertion because of them. She was able to carry on her work for the first three or four hours of an attack, but then generally felt she had to lie down, and if the attack was long, felt tired for a few days. Between the attacks she was able to lead a normal life.

On examination, nothing abnormal could be found. She was thin, but not more than usual. Her heart was not enlarged (12·0 cm. maximum transverse diameter) and the shape was normal. No murmurs were heard and the blood pressure was 130/80. At frequent examinations during the years 1932–1939 no change was observed. Her electrocardiogram was a little unusual with sharply pointed P_2, a notched upstroke of R in leads II and III, and some tendency to right axis deviation; part of the S–T interval was below the isoelectric line, but there was no inversion of the T waves. All these features were constant in many cardiograms taken between 1932–1938 (Fig. 2, A and B).

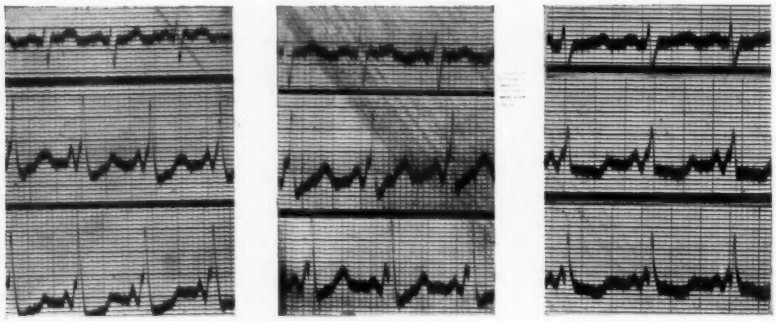


FIG. 2.—Increased inversion of the S–T period in leads II and III after a paroxysm of tachycardia.

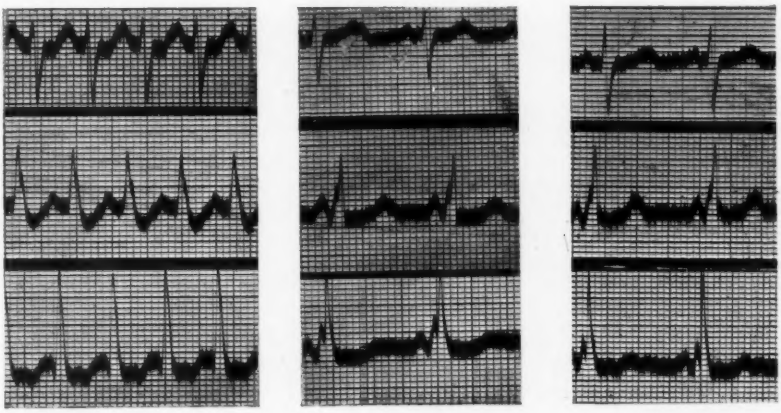
(A) Normal for this patient (7/12/32).
(B) Normal, later (16/11/34).
(C) Some partial inversion of S–T in leads II and III, 5 days after a paroxysm (29/9/38).

In 1935, after an attack lasting six days, she started taking quinidine, 3 grains b.i.d., and had no attack for four months, and then one during each of the next three months, lasting 36, 48, and 17 hours.

She was seen again in September, 1938, having kept an exact record of her four attacks in seven months as follows: March, one of 5 days, 14 hours; June, one of 4 days, 1 hour; August, one of 5 days, 23 hours; and September, one of 5 days, 22 hours. The year had been unusual in the number of long attacks and the absence of shorter ones. Quinidine, as taken in small doses at home, did not appear of value in stopping the attacks, and the absence of short attacks was not due to her taking it regularly, though this seemed to have helped at first.

There was no increase in the size of the heart since 1932, no change of murmurs, and no rise of blood pressure, but the lower T waves in leads II and III (see Fig. 2C) in a cardiogram taken five days after this attack had stopped, made me decide to try to get records just after an attack in which she had taken no digitalis or quinidine, to see if myocardial change was developing or if this was another example of T wave changes after a paroxysm.

On October 11 she came to hospital after an attack which had lasted 8 hours; it was recorded (Fig. 3A) and stopped 17 minutes after she had been given



A B C

FIG. 3.—Showing the absence of anything more than minor changes after a shorter paroxysm in the same patient.

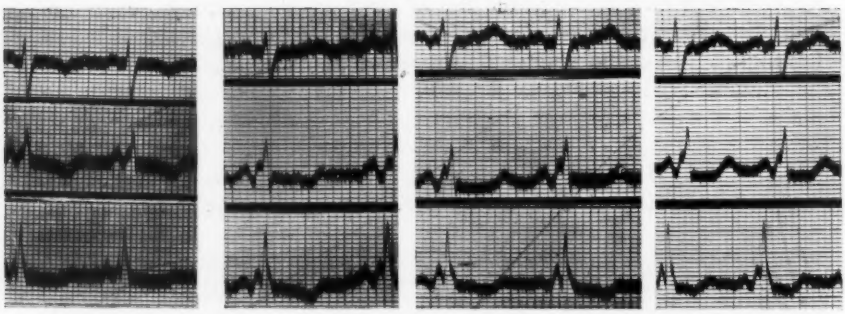
(A) Paroxysm of ten hours (11/10/38).
(B) Slight diminution of the voltage of S–T in leads II and III, two days later (13/10/38).
(C) Practical return to normal four days later (17/10/38).

1·5 mg. of prostigmin intramuscularly. A cardiogram taken two days later showed no change in the T waves except that more of S–T_3 was below the isoelectric level (Fig. 3B); after another four days this was back nearer to her normal (Fig. 3C).

On December 19, the day after an attack which had lasted six days and stopped 45 minutes after her second dose of 5 grains of quinidine (the first being two hours before), there was inversion of T in all leads, especially T_1 and T_2 and T_1 was almost cove-shaped (Fig. 4A). Three days later, T_1 was becoming upright, but T_2 was still inverted and T_3 was more inverted (Fig. 4B). A week later, and still more two weeks later (Fig. 4, C and D), the T waves had returned practically to her normal.

This attack seemed to her just the same as those that she had had for many years and her condition was unchanged after it.

These two patients seem specially good examples of the condition described, in that they have been under observation so many years with their paroxysms without any evidence of other heart disease, and it seems most unlikely that



A B C D

FIG. 4.—Inversion of the T waves in all leads in the same patient after a long paroxysm.

(A) Deep inversion of T in leads I and II, one day after a paroxysm that had lasted six days (19/12/38).

(B) Disappearance of the inversion of T in lead I, but increase in the inversion of T in lead III, T in lead II remaining mainly inverted as before (22/12/38).

(C) Complete recovery of T wave in lead I and disappearance of most of the inversion of S–T in leads II and III (29/12/38).

(D) Further return towards normal (5/1/39).

anything recorded after these paroxysms was exceptional for these patients, and that therefore the T wave changes were the result of the length and severity of the paroxysms, and were not due to any acute organic disease of the heart muscle.

Third Case. I am indebted to Dr. John Parkinson for the notes and electrocardiograms of the following case. Although the details are not complete and it was not possible to follow him for long, it seemed worth including, because the changes were so striking and also because they were noted after three separate attacks.

D.S., aged 21, was in three hospitals for at least six weeks with a diagnosis of chronic myocarditis; no details are available as to why this diagnosis was made.

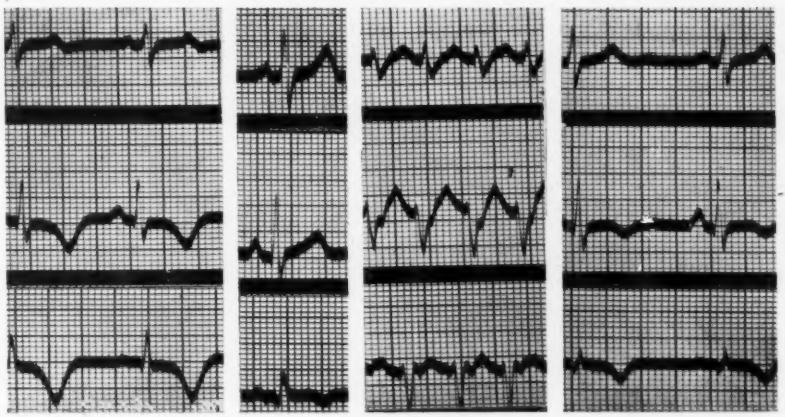
On January 12, 1940, after a month's sick leave, he returned to an R.A.F. station and at once reported sick. His pulse was almost imperceptible at the wrist, and the heart rate at the apex was 174. He said that the condition had started when he was on leave; that he felt breathless and was himself conscious of the rapid beating of the heart; that since the previous October he had suffered from five similar attacks; that all had started suddenly; and that the longest had lasted seven days.

He did not look ill in spite of the attack. Nothing abnormal was found on examination except that the blood pressure was 100/70. A cardiogram confirmed the diagnosis of paroxysmal tachycardia, and as the usual methods failed to stop it, he was given 3 grains of quinidine, after which the heart rate soon dropped to 80. Fig. 5A, taken the next day, showed deeply inverted T waves in leads II and III, such as one might expect after a large cardiac infarct or some other serious myocardial infection. But his age, the absence of symptoms, the quick recovery, and the recurrence of similar changes after two other attacks, all make this extremely unlikely. Within ten days this deep

inversion of T had completely disappeared in lead II, and almost completely in lead III (Fig. 5B).

Another attack started the next day and lasted two days. He was given 33 grains of quinidine and the attack stopped; a cardiogram taken two days later again showed inversion of the T waves in leads II and III.

Nine days later there was a third observed attack (Fig. 5C), which stopped during his sleep. Although this attack had not lasted long, Fig. 5D, taken on the third day, again showed inversion of T in leads II and III: though this was



A B C D

FIG. 5.—" Coronary " T wave changes in leads II and III after paroxysms of tachycardia in a healthy young man.

(A) Cardiogram with deeply inverted T in leads II and III, taken one day after a long paroxysm of tachycardia had stopped. Only one dose of 3 grains of quinidine had been taken (16/1/40).
(B) Return to normal, ten days later (26/1/40).
(C) Cardiogram ten days later (5/2/40) of another paroxysm which had stopped spontaneously during the first night.
(D) Recurrent inversion of T in leads II and III, in cardiogram taken on the third day after another attack (8/2/40).

not nearly as deep as after the longer attack (Fig. 5A), possibly because it was after a shorter attack, possibly because it was taken three days instead of one day after, or more probably for both these reasons.

A fourth attack was observed a few days later, after which he remained quite fit and free from any attack for a month.

DISCUSSION

Five cases, two reported previously and three given in more detail here, have been recorded ; in all striking changes in the T waves after paroxysms of tachycardia were suggestive of progressive coronary disease such as cardiac infarction. The first two ultimately died and though all the evidence seemed against it, it may be that they really had organic changes underlying these electrocardiographic changes. It was, therefore, decided to get records in all

possible patients during the period after their paroxysms of tachycardia, and the first two cases, here reported, were two of the first instances found with such T wave changes; in one they had been suspected previously from earlier records, and in the other there was no previous evidence. It was intended to continue the collection of an additional number of cases to obtain some even more satisfactory records, but the onset of the war has prevented me keeping in touch with the necessary number of patients with paroxysms, and as anyone who is working at the subject will realize with such an erratic condition it always takes a long time to get any particular type of observation that one requires. I was, therefore, most grateful to Dr. Parkinson for the last case, for although alone it might not be very convincing owing to the incompleteness of the history and the relatively short time that has passed since the cardiograms were taken, he was an apparently fit young man without other symptoms and the T wave changes after more than one attack were so striking.

It is probable that cardiologists have been generally familiar with the occurrence of such T wave changes after paroxysms, but that they have not obtained enough of such records for publication. Paul White (1941) in a recent article on inversion of the T wave in lead II in various conditions without organic heart disease did not refer to such cases, and it seems, therefore, worth putting on record, partly because of its scientific interest, and partly because of the practical importance that such patients should not be wrongly diagnosed as having organic heart disease because of the temporary T wave changes.

I have no evidence to bring forward as to how these changes are produced. It may be that chemical or other changes in the myocardium as a result of a prolonged attack, are responsible, and in this sense the changes would, to some extent, be a measure as to the severity and seriousness of the attack, and might indicate the need for adequate rest. Another possible explanation is that it depends on changes in the position of the heart, and that as the diaphragm sinks, or as the stomachs empties, the heart returns to its more normal position with the return to its normal electrocardiographic pattern. Although this would bring the cases into line with some of the other T wave changes that have been reported, and admittedly the third of these cases had a mobile heart where changes did influence the electrocardiogram, this explanation seems most unlikely, for one reason because the time taken for recovering from these changes is measured in days rather than hours.

Summary

After long paroxysms of tachycardia the T waves may become inverted in one or more leads for some days. This does not indicate any organic disease, but is a completely reversible process indicating some degree of exhaustion or strain of the heart muscle.

References

Campbell, M. (1939). *Brit. Heart J.*, **1**, 123.
White, P. D., Chamberlain, F. L., and Graybiel, A. (1941). *Ibid.*, **3**, 233.

PRINTED IN GREAT BRITAIN BY WILLIAM CLOWES AND SONS, LTD., LONDON AND BECCLES.